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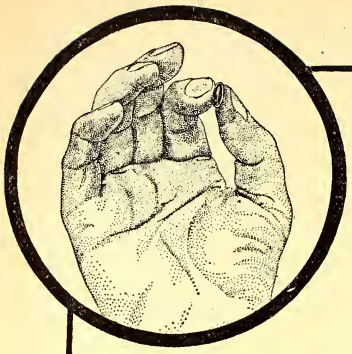

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ORIGINAL ARTICLES.

VACCINE THERAPY IN COLON-BACILLUS INFECTION OF THE URINARY TRACT.[1]

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Colon bacilluria occurs in fully 50 per cent. of all cases of bacteriuria. The condition may be unattended with perceptible effect, either local or systemic. Patients may suffer from dysuria with frequent urination and the colon bacilluria may be the only recognized morbid condition. Usually the bladder irritation is ascribed to the hyperacid urine, but it may continue when the urine is rendered alkaline. That the colon infection is the chief cause of the bladder irritation is presumptively proved by the relief of all symptoms coincident with the disappearance of the bacteria from the urine.

Colon bacilluria may be present with recognizable morbid changes in the urinary tract; the bacteria are either the cause or are closely related to the disease process. The morbid anatomical change, probably, frequently preëxists in the mucous membrane of some portion of the urinary tract. The urethra, prostate, ureter, kidney pelvis, and kidney may be involved. A urinary calculus may preëxist and also may result from a colon bacilluria. Colon urinary infection may be present with tuberculosis of the urinary tract and apparently aggravates the associated morbid anatomy, and intensifies the disturbance of the urinary apparatus and the general symptoms. It may also rarely be present with and aggravate the local disturbance and general symptoms of gonococcic infection of the deeper urinary

[1] Read at a meeting of the Medical Society of the State of New York, January 26, 1910.

tract; of pyogenic streptococcic and staphylococcic, proteus, influenzal, typhoid-bacillus, and other bacterial infections of the bladder and kidney pelvis. Prostatitis, cystitis, ureteritis, pyelitis, and pyelonephritis may occur with colon bacilluria alone and as a mixed infection, especially in tuberculosis of the urinary tract. Chronic arthritis, myocardial degeneration, myalgias, and various other systemic conditions apparently may be related to the urinary infection.

Mode of Entrance of Colon Bacilli into the Urinary Tract. This may be through the urethra in the female with or without instrumentation, and in the male probably only by instrumentation. The prevalence of colon bacilluria in people who have never had a catheter or sound passed into the ureter, proves the existence of other routes of infection. In the vast majority of patients the source is unquestionably the gastro-intestinal tract. Obstinate constipation or diarrhea, attended with more or less injury of the intestinal mucosa, renders the intestinal wall previous to the bacteria, which may then be carried by the blood or lymph stream to the kidney, ureter, and bladder. Colon bacilli from this source have been proved to take on more virulent characteristics.

The diagnosis of bacteriuria is easily made by microscopic examination. The character of the bacteria usually requires a cultural examination of the urine. From a, preferably catheterized, specimen, primary plate cultures should be made; the final recognition of the bacterium by subcultural and tinctorial tests is a common laboratory procedure. A careful physical examination of the patient, with chemical and microscopic study of the urine, will enable one usually to make an anatomical diagnosis. One should never fail to make a careful examination of the external genitals of the patient, both male and female, for focal infection. The rectum should also be inspected. The prostate should be palpated, and possible sacculation of the bladder by abnormal deviations of the uterus and by a lax vaginal wall should be investigated. If indicated, a cystoscopic examination and catheterization of the ureters should be made. The greatest care must be exercised to catheterize the ureters. This is especially true when the bladder is badly infected. The anatomical diagnosis is most important from the therapeutic point of view. If a morbid condition of tissue exist which interferes with the function of the urinary apparatus, no permanent benefit will result from medical treatment, until as nearly as possible a normal anatomical condition is brought about. Colon bacilluria may not be removed as long as poor drainage of the urinary tract exists because of sacculation of the bladder, enlarged prostate, stricture, pressure obstruction, or kink of the ureter, kidney-pelvis sacculation, or if a calculus or other foreign body be present.

Formerly the recognized treatment of colon bacilluria consisted preferably in prolonged rest in bed, a copious liquid diet of milk,

soups, broths, etc., and the use of urinary antiseptics—of which hexamethylenamine is the best. By this method treatment was long, extended to months, and the result was often poor. For the last five years in the medical clinic of Rush Medical College associated with the medical wards of the Presbyterian Hospital and the laboratory of the Memorial Institute for Infectious Diseases, bacteriuria has been carefully studied and many patients have been treated with autogenous vaccines. The work has been carried on by the clinical department of the college and hospital. I have received most valuable coöperation and aid from my colleagues and assistants. The bacterial cultures and autogenous vaccines have been made chiefly by Dr. D. J. Davis,[2] of the medical department, and now an assistant in the Memorial Institute for Infectious Diseases.

When possible the agglutination, opsonic index, bacteriolysis, and the leukocytic blood reaction were studied in each patient. The observation of other reporters as to the character of the bacteriuria has been confirmed. Those suffering from infection of the urinary tract due to the colon organism comprise more than 50 per cent. of the patients with urinary infection. Frequently the gonococcus was obtained with the colon infection from the prostate or seminal vesicles by stripping those organs with the finger in the rectum. Undefined bacteria were sometimes found with the colon; occasionally the unknown organism would be obtained only in plate cultures, failing to grow in anërobic or aërobic subcultures. In some instances the unknown bacterium persisted in the urine after the colon bacillus had disappeared and the patient was symptom-free.

Colon-bacillus infection with tuberculosis of the urinary tract occurred in two patients; the great discomfort occasioned by bladder pain, frequent urination, and septic fever was almost entirely relieved by the disappearance of the colon infection after autogenous vaccination. Two patients suffering from essential hematuria with colon infection have been treated by vaccination. In one, a woman aged twenty-four years, intermittent hematuria had existed for six years or more. A moderate pyuria existed. Repeated examination of the urinary sediment failed to reveal tubercle bacilli. Animal inoculation with the urinary sediment was negative. The ophthalmo-tuberculin test was negative. Cystoscopic examination revealed a normal bladder mucosa. The ureteral catheter entered the right ureter with difficulty and the drop by drop fluid obtained contained blood, leukocytes, and colon bacilli. The left ureter was normal and the freely flowing urine was practically normal. In June, 1907, a right nephrotomy was made; the urine from the kidney pelvis contained red cells and leukocytes and

[2] See report by Dr. David John Davis, "Immune Bodies in Urinary Infections with Colon Bacilli and Treatment by Inoculation," Journal of Infectious Diseases, 1909, vi, 224.

the colon bacillus. The mucosa of the pelvis was thickened and congested. The kidney capsule stripped off normally and a section of the cortex showed histologically interstitial diffuse nephritis. The kidney pelvis was packed with gauze and later was daily injected with argyrol solution, which penetrated to the bladder. Hemorrhages recurred before the external wound had healed and afterward. Six months later, in January, 1908, the patient was again taken into the hospital and injections of autogenous colon vaccines were given every seven to ten days until April, 1908. Hemorrhage ceased. Since that time the urine is blood-free except for a few red cells in the centrifuged sediment. No urinary symptoms remain.

A physician of fifty-eight who always has enjoyed good health, suffered from hematuria without pain in August, 1909. In October cystoscopy revealed a normal bladder mucosa, bloody urine flowing from the right ureteral orifice, and normal urine from the left ureter. Ureteral catheterization was negative for obstruction or stone and the *x*-rays also were negative. Probable tumor of the kidney was diagnosticated. Later he was admitted to the Presbyterian Hospital. The physical examination revealed a good general condition. The urine contained much free blood, many leukocytes, no casts, and was acid in reaction. A pure culture of colon bacilli was obtained from the urine. A milky fluid obtained by stripping the prostate showed many pus cells and a few Gram-negative intracellular biscuit-shaped diplococci. The prostate was stripped every three or four days until no discharge was obtained. The patient was treated with the autogenous colon vaccine every seven days. The blood disappeared from the urine after the third vaccination. The urine remains blood-free and the patient is apparently well.

In September 19, 1908, a physician, aged twenty-nine years, was seized with anuria, and uremic convulsions, which were partially relieved the first day. Headache, vomiting, occasional mild convulsions continued for six days. The scant urine contained a good deal of pus, but no casts or blood. October 21, 1908, he was admitted to the Presbyterian Hospital. The general examination revealed no perceptible morbid condition of heart, bloodvessels, lungs, or abdominal organs. The arterial tension was 120 mm. The eye-grounds were normal. The urine was acid, contained many polynuclear leukocytes (60 per c.mm. of urine), no casts, no red cells, and a trace of nucleoalbumin. Many bacilli were seen and a pure culture of colon bacilli was obtained. The history revealed the probability that the colon infection of the bladder had existed for five years. During that time albuminuria was present for two years and thereafter occasionally only. A month preceding the convulsions he was conscious of lessened strength and endurance, dull headaches, anorexia, and lessened excretion of urine. Autogenous colon vaccination was begun with 400,000,000 bacteria on November 11, 1908. These were repeated every seven to ten days until December 11, 1908, at which

time the urine was almost free of bacteria and pus cells. The patient continued the treatment at home. On March 23, 1909, the urine was sterile and pus-free. The patient has had no relapse.

A man aged thirty-one years, suffering from tuberculosis of the urinary tract which began in the right testis in 1903, was admitted to the Presbyterian Hospital in October, 1907. The right testis had been removed in 1903 and a right nephrotomy and curettage of the kidney pelvis was done in June, 1907. The patient was suffering greatly, although constantly narcotized with opium. There was a septic temperature. No perceptible evidence of tuberculosis of lungs or lymph glands was present. The urine was very cloudy and discolored with abundant pus, blood, and bacteria. Tubercle bacilli were abundant in the urine sediment. Per rectum a nodule in the right lobe of the prostate was tender. The right ureter could be felt as a thick tube, and this and the resistant bladder wall were very tender. From the urine was obtained a pure culture of the colon bacillus. The patient was given absolute rest in bed, and received tuberculin (N.T.), 0.001 mg., every seven or eight days and coincidently therewith was vaccinated with 500,000,000 autogenous colon bacilli. With the third injection the urine became colon-free. Coincidentally the urine became much clearer, containing less pus and blood. The frequency of urination lessened from every one-fourth to one-half hour to as long as three or four hours. The general condition improved by the disappearance of fever and sweats and the appetite returned. Opiates were discarded. The patient left the hospital in December, 1907, and has remained on a farm. He has continued to use the injections of (N.T.) tuberculin, 0.001 mg., every seven to ten days. Examination of the urine every six months reveals the presence of a few leukocytes, red cells, and small clumps of tubercle bacilli. The bladder irritation is not severe and the general health is good. Probably recovery would occur if the patient could take prolonged rest.

These case reports suffice to illustrate the utility of colon vaccine therapy. In a later paper on vaccine therapy in bacteriuria a detailed tabulated report will be made. Patients suffering with pyelitis with colon bacillus infection have recovered with autogenous vaccination when there was no obstruction to drainage. Improvement may occur under the treatment in all cases, but entire recovery from the colon bacilluria will usually not occur if there is stagnation anywhere in the urinary tract. If the enlarged prostate is at fault, rational massage of that organ may be all that is necessary. If there be deformity of the pelvic organs or distortion of the kidney pelvis, or the existence of a urinary calculus, surgical interference should be instituted.

Systemic effects of urinary focal infection must not be overlooked. A chronic infectious arthritis, myocardial degeneration, so-called chronic muscular rheumatism, and neuritis may be related to the

urinary infection. The resistant epithelial layer of the urinary tract probably prevents toxemia until long continuation of the infection causes injury of the epithelial layers and then absorption of toxins may occur.

The bacillus isolated from cases of colon bacilluria differ from each other more or less in size, luxuriance of growth, etc. It would seem rational, therefore, to use autogenous vaccines. This is easily done. Cultures may be made from the urine after it has been transported a thousand miles to a laboratory, by one properly trained in bacteriological technique. We have had no experience with commercial stock vaccines and no comparison may be made of them here.

The autogenous vaccine may be made by heating the culture to 60° C. for thirty minutes. This has proved to kill the bacilli, as shown by control cultures. Fresh suspensions of the dead bacilli should be used. Suspensions more than two weeks old may not give the same results. Usually the first vaccination is made with 200,000,000 bacilli. The subsequent dosage may be gradually increased until a decided local and general reaction occurs. The maximum dose in our work was 1,000,000,000. Experience has proved that smaller doses are preferable to large ones with some patients; 5,000,000 to 100,000,000 may produce sufficient reaction for curative purposes and diminish the risk of a too great reaction. Absolute rest, much of the time in bed, with a copious fluid diet, chiefly milk, shortens the course of treatment, reduces the risk of chill with the reaction, and makes recovery more certain.

Specificity of Vaccine Therapy. The specific effect of autogenous colon-bacillus vaccine therapy is proved by the phenomena of reaction. This consists of the local reaction at the point of injection, which includes redness of the skin, tenderness, and swelling over an area from one to two inches square. This begins in one or two hours after the injection, reached the maximum in twelve to eighteen hours and gradually disappears by the end of forty-eight to seventy-two hours. A general reaction occurs in two to twelve hours, manifested by general malaise, aching of muscles, bones, and joints, more or less headache, more or less fever, sometimes preceded by a chill and leukocytosis. If the patient is up and about reaction is more severe—manifested by severe chill and fever. In many patients there is irritation manifested by pain, aching, etc., of the kidney, bladder, joint, group of muscles, etc., respectively, which is the seat of morbid change due to the colon infection. The specificity is further indicated by an increase in the opsonic index, and finally by an immunity manifested by the failure of reaction after vaccination and the disappearance of the bacteria from the urine. One should employ at the same time all rational measures to relieve the patient. General hygiene, personal cleanliness, correction of diarrhea or constipation, hematinics when necessary, and, as stated

above, surgical or mechanical measures to correct anatomical faults which interfere with proper drainage of the urinary tract.

Elsewhere in the paper I have stated that colon bacilluria is not an uncommon occurrence. In many individuals with this urinary infection there may be no perceptible effects from it. In other patients who suffer from some systemic infection, the conditions may be ascribed to the existing colon bacilluria without due regard for some other possible cause. This statement I think is necessary, because I have found that colon infection of the urine has been brought into the foreground by some physicians who have known of pathological effects due to it and who may misinterpret the condition and fail to look for or to find a real focal infection somewhere else in the body. We must not forget that focal infection of the tonsils, of the sinuses of the head, or of some other mucous tract of the body may produce systemic disease. Therefore, while I believe that colon bacillus infection of the urinary tract is sometimes a cause of not only local but also of systemic disease, I would caution those who find this infection of the urine not to be led astray by it, and to make sure of its relation to local or systemic evidence of disease by proof of its specificity by agglutinative, phagocytic, bacteriolytic, and other tests, and at the same time to look for other possible sources of infection before the treatment is begun.

PAROXYSMAL ARTERIOSPASM WITH HYPERTENSION IN THE GASTRIC CRISES OF TABES.

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Among the most interesting of the problems which the clinician has to solve is that of the interpretation of acute abdominal pain. The subject is a large one, and it is my purpose in this communication to deal with only one phase of it—that indicated by the title of this paper. The topic may best be approached by the presentation of a case which illustrates the main features of the condition under discussion.

Hattie T., aged forty-nine years, white, married woman, a cigar-maker, was admitted to Ward G, Johns Hopkins Hospital, October 9, 1909.

Her complaint was severe pain in the back and stomach, and headache. Her family history is negative. She has been married twenty-four years. She is the mother of six children, two of whom died in infancy. She had had several miscarriages, all occurring early in the pregnancies.

Except for vague "rheumatic" pains for some ten years she suffered from no disease until the present trouble began. About eight years ago she began to have attacks of pain in the back, indigestion, and pains in the joints, especially in the shoulders and knees. The pain in the small of the back was tolerably severe, and was sometimes associated with nausea. These attacks recurred at intervals. One and one-half years ago she separated from her husband, and since then the attacks have increased in number and severity. In May, 1909, the uterus, tubes, and ovaries were removed in one of the city hospitals, but since the operation she has been more nervous than before. She has suffered much from headache, frontal and vertical, and she thinks her eyesight has failed during the last few years.

The individual attacks begin with a feeling of a lump in the throat which cannot be swallowed (globus?). Vomiting soon comes on, so that nothing can be retained in her stomach, and she has extreme pain in the back and abdomen and complains of sensitiveness of the skin of the trunk. The pain is so severe that she usually weeps violently and tosses about in bed, grinding her teeth. She has never lost consciousness in an attack, nor has there been any disturbance of the sphincters. Her mind seems clear during the attacks. Her physician frequently was compelled to give her morphine for the pain. She has had more or less of the drug during the last five or six years. Since about a year ago there have been nearly two attacks per week, each lasting from a few hours to three days. She has noticed palpitation of the heart during some of the attacks.

On examination the patient was found to be somewhat emaciated; the skin was sallow, the muscles soft and flabby. There was no anemia. Her eyes were rather prominent; there was a tendency of the eyeball to run ahead of the lid in making von Graefe's test. The pupils were contracted, and reacted but little to light or accommodation, but it was thought on admission that this might be due to the morphine. There was no glandular enlargement. There was slight enlargement of the heart, the relative dulness extending to the left 10 cm. from the mid-sternal line. The radial and temporal arteries were tortuous and somewhat thickened. The lungs were negative, except for a moderate grade of emphysema. The stools contained some mucus, but no parasites or blood.

On the day after admission she began to suffer from severe pain in the abdomen and back, lying in a crouched position, crying constantly, and complaining bitterly. She vomited at short intervals. The vomitus was greenish in color and was accompanied by nausea. Chemical examination showed a total acidity of 42 per cent.; 23 per cent. of free hydrochloric acid; no lactic acid; no blood. Examination of the blood revealed: Red blood corpuscles, 4,258,000; white corpuscles, 13,800; hemoglobin, 92 per cent. Differential count: Polynuclears, 66 per cent.; large mononuclears, 7 per cent.; lymphocytes, 24 per cent.; and eosinophiles, 3 per cent.

An examination of the stool two days later revealed the presence of ova of Trichocephalus dispar and also ova of Ascaris lumbricoides.

On October 13 I observed her myself during a paroxysm of pain. The face was very anxious, the lips somewhat cyanotic, the eyes reddened and lacrymose. One got the impression at once that the pain was that of organic disease. The radial pulse was 124, regular but of very high tension, feeling like a fine whipcord under the finger. The blood pressure was measured at once and found to be about 190 mm. Hg. She was given an inhalation of amyl nitrite, and the pressure fell at once to 90. A short while after, however, the pressure again became high, going to 200 and later on to 210 mm. Hg. The knee-jerks were overactive; the plantar reflexes normal. The pupils did not respond to light. There was no tactile anesthesia of the chest, but definite analgesia in large areas in the lower extremities were present.

The urine contained no albumin or casts. Acetone was present, doubtless due to the prolonged vomiting, though the test for diacetic acid was negative. Palpation of the abdomen revealed nothing abnormal.

In spite of the active knee-kicks, I felt that the character of the pain and the vomiting, together with the sluggish pupils and the analgesia of the legs, made the diagnosis of gastric crises of tabes probable. This diagnosis received support also from the extreme hypertension due to arteriospasm accompanying the attack. I suggested that lumbar puncture be done and the spinal fluid examined. On the same day Dr. Kingsley withdrew 10 c c. of cerebrospinal fluid. It was under a pressure of from 150 to 200 mm.. H_2O, clear and colorless. There were 50 cells per cubic millimeter, all lymphocytes. The fluid contained both globulin and serum albumin. These tests demonstrated the existence of either a luetic or a metaluetic lesion of the central nervous system.

Sensation was carefully tested on October 15, when the left lower extremity was found to be almost wholly analgesic and the right also, except for a portion on the lateral surface of the limb. There was also analgesia in the domain of the second thoracic of each arm. A patch of analgesia was found upon the right side of the scalp (Figs. 1 and 2). Touch was nowhere impaired and thermal sensation was not markedly involved.

On October 20 the eyes were thoroughly examined by Dr. Bordley. One of them had been dilated with atropine. The other showed extreme myosis and did not react to light and only imperfectly to accommodation. Stelwag's and von Graefe's signs were positive. Convergence was poor. There was advanced arteriosclerosis of the retinal vessels, some of the smaller arteries being almost completely obliterated. The veins were markedly indented by the arteries, and in places tortuous. There was no change in the papillæ nervi optici except hyperemia from obstruction to the venous circulation.

The Wassermann reaction done by Dr. Guthrie was found to be negative.

Examination of the urine: Normal in color; specific gravity, 1010 to 1018; acid; no sugar; no albumin. Microscopic examination was negative. Acetone was present only during the vomiting.

The course of the blood pressure is shown in the accompanying chart (Fig. 3).

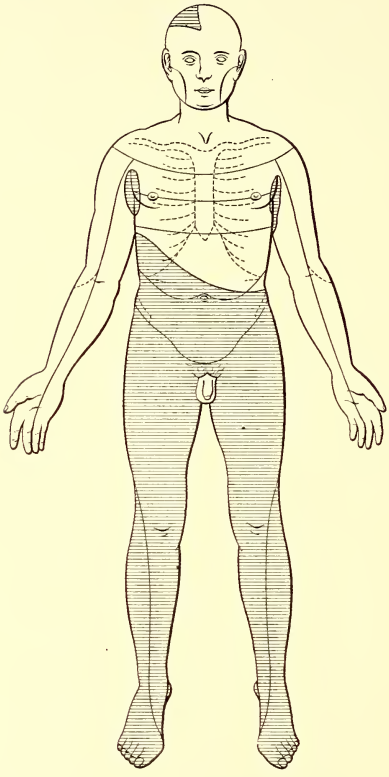

Fig. 1.—Analgesia at the first examination.

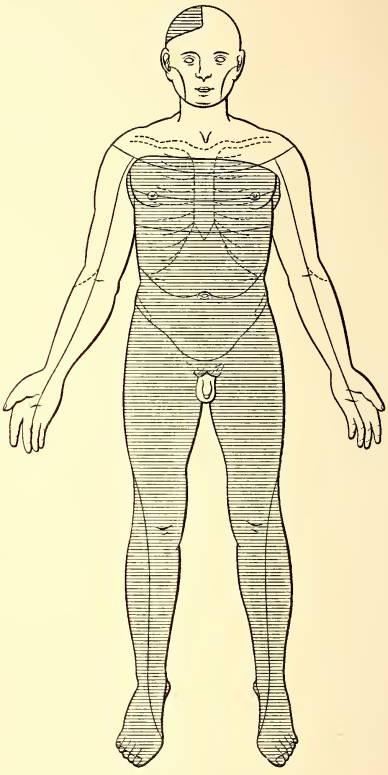

Fig. 2.—Analgesia at the second examination.

The patient's pain was relieved by morphine. As soon as the vomiting stopped she was given small quantities of milk every two hours. During the next five days she had only two attacks of nausea and vomiting. She began to have a good appetite and to feel very much better. The blood pressure (maximal) varied between 175 and 215 mm. Hg. until the 18th. On the 19th the maximal pressure was found to be only 120 mm. Hg., and since then it has varied between 110 and 125 mm. Hg.

Since the fundamental studies of Fournier upon the phenomena of early tabes the pains in the upper abdomen in this disease have

been classified under four main headings: (1) Crises in which there is vomiting alone; (2) crises in which there is pain alone; (3) the *grande crise gastrique*, in which the phenomena are complicated and violent, and include extreme pain, vomiting, and retching, with severe general symptoms; and (4) crises in which the appetite is entirely lost, though other signs may be absent.

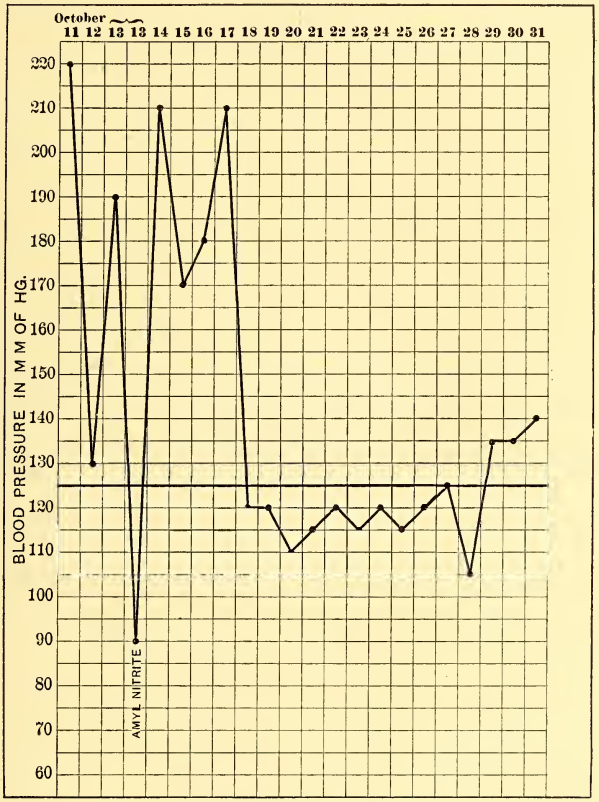

Fig. 3.—The course of the blood pressure.

The patient whose history has been given evidently suffered from crises of the third type, and it is to this form of gastric crises in tabes that I desire to refer, making it, however, distinctly understood that in the other three types of crises many of the features of Type 3 may be lacking.

In the crises from which this woman suffered the pain was situated in the upper abdomen and radiated into the back. The pain was accompanied by paroxysmal arteriospasm, with great elevation of the maximal arterial pressure. That the hypertension depended upon the arteriospasm was evident from the effect of amyl nitrite,

which reduced the maximal pressure promptly to 90 mm. Hg., though as soon as the effects of the nitrite had worn off the hypertension reappeared. The marked oscillations in the maximal pressure during the crises are evident in the blood pressure chart (Fig. 3). It was only after the pressure returned to normal and remained on the normal level that the symptoms disappeared. A study of similar cases in the literature indicates that partial falls of the pressure are significant only of remissions in the crises, not of termination. The abrupt terminal fall in pressure is striking, and the maintenance of a tolerably steady pressure at the low level after the period of hypertension seems to me most interesting.

There are at least three conditions in which attacks of severe abdominal pain with paroxysmal hypertension occur: (1) The gastric crises referred to above; (2) lead colic; and (3) the angina abdominis of arteriosclerosis. A number of cases of all three conditions have been collected and carefully analyzed by J. Pal.[1] In these cases, besides the pain and hypertension, the attacks presented several other characteristic features, including (1) constipation, (2) boat-shaped retraction of the abdomen, (3) in some cases meteorism, and (4) in many instances segmental sensory disturbances (usually hyperesthesia or hyperalgesia) in the root domains of the lower thoracic and upper lumbar spinal nerves.

There has been much dispute as to the origin of the pain in these cases and its relation to the hypertension. Some authors assume a primary neuralgic pain with secondary hypertension due to the pain; others, with Pal, regard the hypertension as the result of vasoconstriction of the small arteries of the stomach and intestines, and look upon the pain as due to stretching of nerves in the arterial sheaths of the same arteries proximal to their constricted portions, assuming that in these proximal regions of the gastro-intestinal arteries the arterial wall is distended and under very high pressure. The researches of experimental physiologists and surgeons tend to confirm the view that the only pain nerves in the stomach and intestines are those in the walls of the bloodvessels. It has long been known that the visceral peritoneum (not the parietal) is insensitive, and there is evidence to prove that even violent contusion of the intestine or stomach (such as crushing with Dupuytren's scissors) causes no pain.

In the gastric crises of tabes it is assumed that irritation in either the posterior roots of the spinal nerves or their continuations within the cord leads to a reflex vasomotor constriction which is most extreme in the splanchnic domain. If this explanation is correct we must assume that we have to deal in tabes at times with elective stimulation of posterior root fibers, for when tabetics suffer from lancinating pains in the lower extremities the blood pressure is

[1] Gefässkrisen, Leipzig, 1905, pp. 1 to 275.

usually low and we must assume in such cases a reflex vasodilatation. In the gastric crises of tabes there is paroxysmal arteriospasm and hypertension, and we must assume here a reflex vasoconstriction. It is interesting that lancinating pains and gastric crises rarely occur together in tabes, though their alternation is not uncommon. This disparity in the symptomatology of incipient tabes, pointing to an elective stimulation of the posterior root fibers or their intramedullary continuations, has led me to think of our embryological knowledge of the posterior roots. Since the studies of Flechsig and, later, of Trepinski we have known that the fibers of the dorsal funiculi do not become medullated all at once. Definite groups of these fibers receive their myelin at very different periods, and Flechsig has subdivided the fibers into four distinct embryological systems.[2] The fibers of these different systems have different terminations in the cord and in all probability subserve different functions. It has also been shown, through the microscopic study of the spinal cord in cases of tabes, that a very distinct parallelism occurs between the areas degenerated in this disease and the embryological memberment in the fœtal cords. Furthermore, it has been shown that in tabes the sequence in which the several systems suffer may vary. It seems to me highly desirable, therefore, that cases of incipient tabes carefully studied clinically, which, through some intercurrent disease, come to autopsy before degeneration is advanced, should be most closely investigated microscopically. In this way we may hope for gradual enlightenment concerning the functions of the different systems of fibers contained within the dorsal roots of the spinal nerves.

The explanation of the phenomena other than the pain and hypertension in the gastric crises of tabes has also been attempted by various writers. Though the explanations thus far offered leave still much to be desired, opinion at present leans to the view that the vomiting is a reflex vagal phenomenon; that the constipation is due to paralysis of the intestine from ischemia due to the vasoconstriction; that the boat-shaped retraction of the abdomen is to be regarded as a reflex through the motor spinal nerves of the corresponding segments; and that the segmental hyperesthesia is to be thought of as due to "referred sensation" in the sense of Head, resulting from the violent impulses passing along the stretched perivascular sympathetic nerves and reaching the cell bodies (within the spinal ganglia) of the neurones of the lower thoracic and upper lumbar dorsal nerve-roots.

Since in arteriosclerosis attacks of angina abdominis closely resembling those of the grand gastric crises of tabes occur, it might be thought that the attacks in the patient reported above were due to the arteriosclerosis rather than to tabes, but, though the knee-

[2] L. F. Barker, The Nervous System, New York, 1899, p. 424 et seq.

kicks were lively, the pupils were very sluggish to light and, above all, the lymphocyte count in the cerebrospinal fluid was markedly increased, and the protein content of that fluid indicated the existence of a parasyphilitic disease. Moreover, vomiting appears to be less common in the angina abdominis of arteriosclerosis than in the gastric crises of tabes.

To one other point attention should be called, namely, the wide distribution of the analgesia and the great differences in this distribution at different periods. In the absence of disturbances of tactile and thermal sensation such an extensive analgesia could scarcely be due to the tabes. It seems much more probable that this analgesia and the globus of which the patient complained are hysterical manifestations complicating the more serious malady.

Should these severe crises continue in this patient, we shall consider the advisability of cutting intradurally the seventh, eighth, and ninth dorsal nerve roots on both sides of the body (Foerster's operation). In Küttner's case and in that reported by Bruns and Sauerbruch[3] the results were eminently satisfactory.

A STUDY OF FIVE HUNDRED AND FIFTY CASES OF TYPHOID FEVER IN CHILDREN.

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In November, 1903, I read a paper before the Medical and Chirurgical Faculty of Maryland, the subject being a study of 337 cases of enteric fever in children. Now it is my intention to present a further study, by analyzing 213 additional cases, which have been treated during the half decade ended December, 1908.

In this study of 550 cases of typhoid fever treated in the Children's Hospital, District of Columbia, many obstacles, some insurmountable, were encountered. The period over which the investigation extends has been divided into three and a half decades, which seem to conform to the changes of ideas respecting this particular disease, embracing the years 1872 to 1908, inclusive. During the first decade all cases of typhomalarial fever were excluded, because this was then thought to be a distinct disease possessing only a few symptoms similar to those found in enteric fever. During the second decade these were included, because, by common consent, all typhomalarial cases were then recognized as enteric. In the

[3] Operativer Behandlung gastrischer Krisen, Foersterscher Operation, Mittheil. a. d. Grenzgeb. d. Med. u. Chir., 1909, xxi, 173 to 178.

third decade, cases of mixed infection which, in their clinical and pathological phenomena were identical with the typhomalarial diseases of the previous decades, have been incorporated. There might have been justification in swelling the number by including the many cases recorded in the first fifteen years under the headings *infantile remittent, remittent continued,* and *irregular* fevers, because I was then serving as an assistant physician in the hospital, and, in the light of our present knowledge, I now believe that such cases were genuine enteric fever. In spite of the fact that continuous connection with the hospital since 1876 has given me ample opportunities for careful study of those cases, I do not feel justified in changing the diagnosis made by my predecessors.

In the earlier years of practice, I was among the few who did not believe in the immunity of infants and young children to enteric fever. When some advanced the opinion (from which they have fortunately receded) that infants rarely, if ever, had enteric fever, and supported it by their failure to find the intestinal lesions of the disease in a large number of necropsies, I strenuously contended that infants and young children had a disease, which clinically was the analogue of enteric fever in the adult, and shortly thereafter presented to our local society the intestine from an infant showing lesions identical with those found in adults who had died of enteric fever. During the past ten years, I have seen an epidemic in one of our institutions, which spent its force upon infants under one year of age; and during the last five years have seen, in private practice, at least a dozen typical cases, several of which were used to illustrate a lecture on typhoid fever in infants, delivered at the Harvard Medical School in 1907. The clinical phenomena, including direct infection, were those of enteric fever, and I presented a specimen from one of the cases which showed ulceration and perforation of the ileum. Of late all doubts on this point have been dispelled by pediatrists generally. The acceptance of the theory of immunity unquestionably obscured the diagnosis in a number of cases, which might otherwise have added to the interest of this paper. Enteric fever in the child differs in degree only from that in the adult. While the clinical phenomena differ somewhat, the structural changes are identical, regardless of age.

There has been an annual increase in the number of cases treated in the hospital relative to the whole number of patients admitted. This has been about uniform, except in two instances, when it was much greater owing to the prevalence of enteric fever, in epidemic form, in the city.

Season. Of the 550 cases, 420 (76.54 per cent.) were admitted during July, August, September, and October.

Table Showing Cases Admitted by Months.

	Cases.		Cases.
January	18	July	78
February	11	August	153
March	6	September	119
April	11	October	70
May	6	November	38
June	21	December	18

Sex. Two hundred and ninety-six boys and 254 girls were treated, this being about the proportion in adults admitted to general hospitals.

Age. At the organization of the hospital the maximum age of children admitted was fifteen years and the minimum two years. The maximum was gradually lowered until in 1888 it was fixed at twelve years, which accounts for the small number between twelve and fifteen years. There is a decided increase in the number admitted after the fourth year, which may be due to increased susceptibility at the beginning of school-life.

Table Showing Ages.

	Cases.		Cases.
One year	1	Nine years	61
Two years	22	Ten years	72
Three years	29	Eleven years	77
Four years	27	Twelve years	33
Five years	50	Thirteen years	3
Six years	50	Fourteen years	3
Seven years	65	Fifteen years	5
Eight years	49	Not given	2

Mode of Conveyance. It is a singular coincidence that the first case of enteric fever treated was attributed to the eating of oysters. This was in 1872, and yet it was quite twenty-five years thereafter that the oyster was recognized as a carrier of typhoid bacilli. Four cases were attributed to polluted milk, 25 to water, and 43 to contagion. In 478 cases no record was made as to the mode of conveyance. In the 43 cases attributed to contagion there was in every case evidence of direct exposure, in many instances several cases having occurred in the same family. The proof is positive in several cases in which water is mentioned as the medium. These cases came from a locality where enteric fever was epidemic at the time of their admission. The children, as well as many, if not all, of those affected, had drunk the water from a neighboring well, which, upon examination, was found to contain the colon and other bacilli, together with fecal matter.

Morbid Anatomy. In 43 cases the necropsy revealed the characteristic lesions and in addition structural changes in the other organs. In 22 necropsies were not permitted.

Perforation was found in 17 cases, all being of the ileum. In 1 case three perforations were found, in 2 cases two, and in the remainder but one.

Hemorrhage. Twenty-eight deaths resulted from hemorrhage, but in no instance could the bleeding vessel be found.

Spleen. The spleen was almost invariably enlarged in the fatal cases.

Liver. No pathological condition was found in the liver. Abscess of the gall-bladder was found in one of the recent cases.

Kidney. Acute nephritis was present in 8 cases, the condition having been recognized before death.

The respiratory organs were affected in 7 cases, the *circulatory* in 3, the *brain* in 3, the *peritoneum* in 2, and the *bladder* in 1.

Mode of Onset. The disease was recorded as beginning insiduously in 361 cases, with diarrhœa in 17, malaise in 14, chills in 66, suddenly in 81, delirium in 7, cough in 5, vomiting in 8, headache in 6, synovitis, stupor, nausea, and tonsillitis in 2 each, and with coryza, adenitis, arthritis, erythema, sweats, and insomnia in 1 each; and the onset is not mentioned in 24 cases.

Symptoms. *Temperature.* The course of the fever in children is usually of the remittent type, ranging from 103° F. to 105° F., and terminates by lysis. In this series the fever in 279 was remittent, in 24 intermittent, in 3 irregular, and in 2 atypical. The fever terminated by lysis in 241 and by crisis in 19. Posttyphoidal rise was noted in 4 cases and was due to some error in management. There were 11 septic cases that were most pronounced; 4 of these died. Recrudescence was noted in a very small proportion of cases. Chills were recorded in 73 cases, 13 being at the outset.

Rose-spots are not as frequent in children as in adults. They were present in 133 cases only, but, as about 20 per cent. of the cases were negroes, the percentage is not accurate. Sudamina and miliaria are more common in the negro child.

Sweats. More or less sweating at the height of the fever is not uncommon, and in this series it was so profuse in 10 cases as to classify them under the sudoral variety.

Bed sores so seldom occur in children that it was not regarded as important to consider them. Furunculosis occurred seven times.

Circulatory System. The changes presented by the blood differ slightly from those found in the adult. There was 1 case of pericarditis and endocarditis and one of endocarditis. One case of phlebitis of the femoral vein was noted.

Digestive System. Five cases of ulceration of the mouth, one being gangrenous, were recorded. One case of esophagismus of exceeding interest was found. The boy was seven years of age and had a typical, moderately severe, attack of enteric fever. At the height of the disease, in attempting to take drink or nourishment, a tonic spasm of the œsophagus would occur. This necessitated rectal feeding. After a long convalescence the patient recovered. Adenitis and parotitis occurred in 21 cases, most of which suppurated. Pharyngeal symptoms were recorded in 8 cases. Diar-

rhœa was of infrequent occurrence after the first stage, and when present was usually controlled by change of food. Hemorrhage occurred in 54 cases (9.8 per cent.); 27 (50 per cent.) of which died. In 1 case there were three profuse hemorrhages and the child recovered, while in the 27 fatal cases, but one hemorrhage was recorded. Meteorism and tympanites were noted in 14 cases. In 6 the distension was unusually great and caused intense suffering. Abdominal tenderness and gurgling were not as frequently observed as in the ordinary diarrhœa of children. Indeed, I regard the gurgling and tenderness in the right iliac fossa as of little practical clinical value. Of the 550 cases there were 17 (3 per cent.) with perforation, all of which were fatal. The diagnosis of perforation was made in every case within a few hours after its occurrence. In several cases an operation was proposed, but the parents would not permit it. In other cases peritonitis developed so rapidly that an operation was not deemed advisable. Three children were operated on when *in extremis*, and died a few hours thereafter. Enlargement of the spleen was recorded in 149 cases, but undoubtedly greater care in case-recording in the earlier decades would have increased the number. Epistaxis was found in 225 cases (40.9 per cent.), in many of which it was troublesome, and in 1 profuse and fatal.

Pulmonary System. Bronchitis was present in 31 cases in the earlier stages. *Pneumonia* was noted in 15 cases, one of which proved fatal.

Nervous System. Delirium. Children usually bear high temperatures much better than adults, but our statistics show a large percentage (56.18 per cent.) of nervous perturbations attributable to the pyrexia. The amount of fever formed no index of mental disturbances.

Table Showing Type of the Delirium.

	Cases.
Mild	51
Low, muttering	155
Wild	59
Maniacal	8
Hysterical	1
Stupor	12
Coma	1

The distinction drawn between *wild* and *maniacal* delirium is arbitrary. Those classed as wild were thoroughly unaccountable and required restraint; while the maniacal had hallucinations, delusions, and violent tendencies. *Convulsions:* In 16 cases convulsions appeared during the fastigium and not at the onset, as is the case in other infectious diseases in children. All of these cases were fatal. *Neuritis:* Local neuritis during convalescence was observed in 5 cases, all of which recovered. *Hemiplegia* with a fatal termination occurred once. *Post-typhoidal* insanity was

observed during convalescence in 9 cases, which had run a mild course without delirium. They were all due to faulty nutrition and promptly recovered with improvement in general health. I have reported 4 of these cases.[1]

Ear. Otitis media supervened in 15 cases (2.7 per cent.). Suppuration was profuse, but the disease only invaded the mastoid cells in 1 case. Deafness, sometimes profound, was frequently observed during the height of the disease, but always disappeared with the subsidence of the fever.

Renal System. Retention of urine is not often met with in children, but it was mentioned in 2 of these cases. The diazo reaction was applied in 182 cases, with 73 (40.1 per cent.) positive. [The test is made when the child is admitted, and daily thereafter, but the positive reaction is often delayed as late as the third week.] This test was abandoned several years ago. Albuminuria during the febrile stage occurred in 40 cases (7.2 per cent.), but usually disappeared during convalescence. Acute nephritis was noted in 15 cases (2.7 per cent.), 5 being fatal.

Postenteric pyemia infrequently manifests itself by abscesses. At one time there were 3 cases of perirectal abscess in the hospital. No case of multiple abscesses was recorded, but a boil on the head, buttocks, thigh, or back was not uncommon.

Association with Other Diseases. In an institution in which tuberculous diseases prevail so extensively, it is somewhat surprising not to find acute miliary tuberculosis associated with, or directly following, an attack of enteric fever much earlier than 1909. Since then pulmonary tuberculosis has fatally attacked 5 convalescents, which are included in this series. Scarlatina, measles, malaria, and pseudo-membranous pharyngitis and laryngitis also complicated the cases. Cancrum oris occurred in 4 cases, all being fatal.

Varieties of Fever. Typhoid fever in children presents such various modifications in its complex symptomatology that its classification as to degree depends entirely upon the observer. The course might be considered mild, and yet hemorrhage or perforation would cause an unexpected fatal termination; on the other hand, a case may be grave from the initial stage, and when least expected a rapid return to health may take place. It has been our custom to classify as follows:

	Cases.
Mild	264
Moderately severe	132
Severe	142
Irregular	2
Sudoral[2]	10
	550

[1] Trans. Amer. Pediatric Society, viii, 177.

[2] The term sudoral is used to define a condition in which there is profuse sweating during the fastigium.

Relapses. There were 48 relapses (8.7 per cent.), 4 cases having two each, with 1 death. In a case of septic enteric fever death occurred during the relapse. My experience has been that a relapse is of milder type and shorter duration than in the adult. The relapses here noted are true ones, but several spurious relapses have occurred which were of slight significance.

Diagnosis. There have been but few mistakes in diagnosis, the greatest difficulty being to differentiate enteric from estivo-autumnal malarial fever, when the blood examination was negative. However, observation and repeated blood examinations finally settled the question. The Widal test was applied in 283 cases and gave positive results in 130 (49 per cent.) This percentage is a trifle lower than the results of other observers and may have been due to imperfect methods, which were unavoidable, before the establishment of a laboratory in the hospital. During the last five years a positive reaction has been obtained at some time during the course of most cases, but in most cases during convalescence, and in a few tests have invariably been negative. No blood-cultures have been made.

Mortality. There were 65 deaths, a rate of 11.8 per cent. Taking the periods separately, we see the greatest reduction in the last decade and a half, which is unquestionably due to the methods of treatment employed. It must be stated that a number, especially colored children, died within forty-eight hours after admission, and, although they are included in the death-list, yet they might, with propriety, be excluded, which would reduce the mortality considerably.

	Cases.	Deaths.	Mortality. Per cent.
1872–1882	26	8	30.76
1882–1891[3]	59	12	20.33
1892–1903[4]	252	28	11.1
1903–1908	273	17	7.9

Analysis of the fatal cases. There were 32 boys and 33 girls, whose ages ranged as follows: Two years, 2; three years, 3; four years, 4; five years, 8; six years, 8; seven years, 8; eight years, 2; nine years, 6; ten years, 12; eleven years, 11; twelve years, 5; and fourteen years, 2. One was infected by oysters, 2 by milk, 5 by contagion, and 4 by water. The necropsy was made in 40 and revealed the characteristic local and parenchymatous lesions. In 4 subjects who had died of pulmonary tuberculosis, the intestinal lesions had healed. The onset was insidious in 40; by gastroenteric symptoms in 8; by angina in 1; by chills in 3, and suddenly in 11.

[3] It will be seen by the table that no cases are given in 1885. This omission, as well as that of 1897, was owing to the loss of records. In the annual report, 1885, 3 cases are reported, all of which recovered, which will reduce the mortality of the second decade to 19.35 per cent.

[4] In 1897 11 cases were treated and 1 died, which will reduce the mortality in the third decade to 9.27 per cent.

There were rose spots in 9. The fever was remittent in 38, intermittent in 6, atypical in 6, septic in 2, and not stated in 2. Epistaxis was noted in 32, chills in 16, intestinal hemorrhage in 26, perforation in 16, nephritis in 11, convulsions in 11, and pneumonia in 6. The delirium was wild in 28, muttering in 22, maniacal in 1. In 9 the spleen was enlarged. Such complications as bronchitis, pneumonia, peritonitis, cancrum oris, aphonia, gangrenous stomatitis, hemiplegia, pulmonary tuberculosis, mitral disease, and endocarditis, helped to swell the mortality. The variety of the fever was severe in 59, moderately severe in 5, and mild in 1. Thirty-five were treated by hydrotherapy; 3 by antiseptics; 6 by eliminative and antiseptic methods; 10 by antiperiodics; and 8 by antipyretics. Excessive diarrhœa was treated six times; hemorrhage fifteen; nervousness four, and cardiac weakness twenty-seven times.

Treatment. The general management of the cases was of the same character throughout, but the systematic methods of the trained nurse, which superseded the crude ones of the unskilled in the first decade, contributed much to the better results obtained in the second and third periods.

The *diet* which was uniformly liquid during the first three decades, consisted of milk and animal broths, except in 2 cases in which "pudding diet" was noted. I am not familiar with this last named food for enteric cases, nor was I able to ascertain its full meaning, but I suspect that the patients for whom the pudding was ordered were in the convalescent stage.

In the first decade there were twenty-six patients who were treated as follows:

	Cases.
Cold sponging	5
Antiseptic treatment	1
Antiperiodic treatment	20

It will be seen that 20 received quinine. During this period Liebermeister's treatment was in vogue, and I can recall the large doses of quinine given, which had little effect upon the fever, but often irritated the stomach and increased the nervous symptoms.

In the second decade 59 patients received the following treatment:

	Cases.
Cold sponging	23
Cold pack	1
Antiseptic treatment	5
Eliminative and antiseptic treatment	4
Nervous symptoms treated	32
Antiperiodic treatment	30
Antipyretic treatment	16

Daylight was beginning to dawn on the treatment, and the beneficial effects of reducing high temperature by external applications of cold were realized. This change of treatment was not well estab-

lished when that pernicious class of drugs, the so-called antipyretics, was introduced. Antipyrin was administered in 16 cases, 4 of which died. I remember with what pride we gave the synthetical preparations to demonstrate their power of quickly reducing high temperatures. It was quite two years before we realized that, while the temperature was being so beautifully lowered, the necessity for stimulation increased.

Quinine was now given to 50.8 per cent. of the cases and undoubtedly played its part in augmenting the number requiring stimulation.

In the third decade the 252 cases were treated as follows:

	Cases.
Hydrotherapy	213
Antiseptic treatment	33
Diarrhœa	4
Hemorrhage	1
Heart stimulants	31
Nervous symptoms	1
Antiperiodic treatment	27
Antipyretics	1

In this decade the treatment by the various *intestinal antiseptics* was introduced, but, after a fair trial, they were discontinued in my service, because I was not convinced that any benefit resulted from their administration.

Twenty-seven received quinine, because of mixed infection, but only 4 were given antipyretics; phenacetin was given to them to allay nervous manifestations and not to reduce temperature. Of 252 cases, 213 were treated by hydrotherapy. Under this head are included cold sponging, the cold pack, and tubbing after the method of Brand. The *regular* treatment recorded in the tables means one or all of the three methods, according to the indications in each case. About the same number as in the previous decade received stimulants, but for a different purpose. While in the second decade such drugs were necessitated by the cardiac depression from the coal-tar derivatives, now they were given as routine treatment in carrying out the Brand method.

In considering the results obtained in this one hospital during the last decade, it may not be wise to lay too much stress upon figures. The reduction in death-rate may not be due to the treatment, but by comparison with the mortality in preceding periods under different methods it emphasizes the following facts:

From 1892, the beginning of the third decade, to 1898, inclusive, the treatment followed was hydrotherapy, antiseptic, and antiperiodic. During this time 88 cases were treated, 10 of which died, giving a mortality of 11.36 per cent. At the beginning of 1899 the purely hydrotherapeutic treatment was begun and has been strictly followed throughout the remainder of the decade, with the result that of the 164 cases then treated 18 died, giving a death rate of 10.97 per cent.

Four cases of the last series died of pulmonary tuberculosis either during or directly following a typical course of enteric fever. We may rightly exclude them from our mortality list—when the death rate will be 8.54 per cent.

Some slight changes in treatment have been instituted during the last half decade. The diet was augmented in nutritive value by the addition of cereals, eggs, and bread to the prescribed routine of milk and broths. The patient's ability to digest such "soft food" was carefully watched and only two or three instances were recorded in which the semi-solid food had to be discontinued. I was rather skeptical at first as to the good results from giving semi-solid food to typhoid-fever patients, but I must confess to a conversion to the method so well portrayed by Shattuck. Realizing that diarrhœa is the exception in the child with typhoid fever, one can safely venture the use of liberal feeding. While an occasional case may not tolerate anything but liquid food, the majority will not only relish, but will digest and assimilate soft toast, cereals, and soft boiled eggs. By adopting this method, most of the heart-rending scenes of the sick-room will be avoided. The otherwise patient child no longer begs and cries for "something to eat;" there is no longer progressive emaciation until the little skeleton is covered by loose skin hanging in folds: relapses become less frequent; convalescence is shortened; and complete recovery replaces prolonged invalidism.

Our ideas on the Brand method have changed materially in that it was only used in three cases during this last period. Its beneficial effects have been demonstrated, but children do not require such heroic treatment; the sponge bath is quite as effective in reducing temperature, in allaying nervous perturbations, and in stimulating the activity of the emunctories. Intestinal irrigation was recently tried in a few cases, but its beneficial effect is not yet evident.

The table shows the methods of treatment:

	Cases.
Regular	165
Brand	3
Quinine	22
Irrigation	24
Antiseptic	15
Eliminative	3

In concluding, I desire to state that the cases were culled and the charts prepared from many imperfectly kept records, by the assiduous labors of Doctors Grasty, Turner, Riley and Smith in 1903, and by Doctors Ong, Titus, McLaughlin and Durney in 1909. To all of these I acknowledge my indebtedness.

ARTERIAL HYPERTENSION.

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Abnormally high blood pressure, if once permanently established, is a condition of much significance, as it constitutes a grave departure from the physiological norm and entails certain structural and degenerative secondary effects in the arteries and heart of the most profound character. The mere fact of blood pressure being habitually raised above normal, even to a considerable extent above normal, does not seem necessarily to circumscribe the individual's activities, nor is it always accompanied by noteworthy subjective discomfort. The change to high levels is ordinarily a very gradual affair, the tissues progressively accustom themselves to the new standards of pressure, and the economy may not be disturbed. We consequently often find individuals with systolic readings over 200 mm. Hg. actively engaged in business and professional affairs, unconscious of any disturbance of health. Slowly but surely, nevertheless, the excessive mechanical strain to which their circulatory organs is subjected begets serious degenerative alterations, and finally if the process be not stayed the whole apparatus of the circulation will one day fall to pieces like the "Deacon's one hoss shay." Meanwhile no symptoms of alarming nature may arise until a sudden cardiac failure or an apoplexy tragically reveals the true state of affairs. Recently I was consulted by an active business man who for several weeks had unsuccessfully treated a cough, which was attended by asthmatic symptoms. He was found to have a blood pressure of 245 mm. with a pulse of 124, gallop rhythm, jugular pulse, pulmonary congestion, hepatic hyperemia, and albuminuria. In many a man above sixty we find a basic systolic murmur and accentuated aortic second sound indicating hypertension, or we may encounter indications of a relative mitral leak, with no complaint on the part of the patient beyond a certain puffiness on exertion.

Arterial hypertension, except when it occurs in association with chronic nephritis, is comparatively seldom met with before the age of forty. As an accompaniment of chronic nephritis it is often encountered in early life, even during childhood; otherwise it may be considered a disease of maturity. After forty years is passed the older the patient is when hypertension is diagnosed the more pronounced as a rule will be the element of arterial degeneration and the more apparant the cardiac secondaries. In early middle life unless the elevation of blood pressure is the result of nephritis it is apt to stand unique as the single physical indication of some obscure nutritive or toxic disturbance. In many instances of the kind hypertension

is the only thing present without or previous to the development of organic secondaries. Cook advocates the collection of these cases into a separate group for which he suggests the name "essential arterial hypertension." Under this designation he would include that residuum of cases in which after eliminating all the usual and known causes there remains no explanation for the one constant and only physical sign—hypertension of the pulse. In practically all of these cases, as he states, there is a certain element of cardiac hypertrophy, but it may be difficult to recognize. This definition very nearly coincides with the presclerosis of Huchard. I can best describe this type of case by clinical illustration: S. P., aged fifty years, was rejected at a life insurance examination for a policy of large amount, because he had a blood pressure of 165 mm. He consulted me to determine its cause. He was a perfectly healthy looking man of active habits and although a generous liver was not intemperate. He was an excessive smoker. Careful examination failed to reveal any indication of organic disease. His heart may have been hypertrophied, but regarding this, after repeated examinations, I still remained uncertain. The urine was free from albumin and casts and of normal quantity. He complained of flatulency, but was not constipated. His hygiene and diet were carefully regulated, his smoking moderated, and in a year the blood pressure had descended to an average of 135 mm.

This form of primary arterial hypertension is probably more frequent than we are aware, and if the sphygmomanometer were used on all men of middle age coming under observation, both in clinical routine and life insurance examination, attention would be directed to it oftener than is now the case. It is hardly likely that we have to do here with a different type of case from those we ordinarily class as arteriosclerosis with hypertension, or as chronic nephritis. We have encountered it in its incipiency, that is all, before pronounced organic secondaries have developed to stamp the case as arterial, cardiac, or renal. Should the condition persist unmodified we would probably be able to watch the development of arterial fibrosis as the result of prolonged mechanical strain upon the vessels, of cardiac enlargement, casts and albumin, and all the flock of secondaries that characterizes the fully developed disease.

The relationship of arteriosclerosis to high blood pressure still remains difficult to define, notwithstanding the large amount of investigation and discussion devoted to the problem. It is a well known clinical fact that the most extreme degeneration of palpable arteries may exist without elevation of blood pressure above the normal (Dunin, Groedel, Elliott). Equally common in practical experience is the existence of a greatly elevated blood pressure with but slight fibrosis of accessible arteries. We have to account then for the circumstance of an individual with rigid calcareous radials presenting a normal pressure for his age, whereas another patient much younger and possessing vessels far less degenerated gives

pressure values double the normal. It would appear that hardening of the superficial vessels alone does not suffice seriously to disturb the normal circulatory pressure. It is necessary that we distinguish very positively between the clinical type of arteriosclerosis and hypertension. They do not by any means go hand in hand and when the two are found together they may only have an indirect relationship. The fact that in most cases of persistent high pressure some fibrosis of accessible arteries can be made out has probably led us to overestimate the importance of arteriosclerosis in elevating blood pressure. Undoubtedly stiff arteries may cause some increase in tension without the operation of any second factor, but that they can unaided produce the excessive values we deal with clinically is open to question. "Arteriosclerosis is an anatomical change, whereas high pressure is a functional disturbance." Instead of arterial changes giving rise to high pressure it is probable that as often as not when the two are found together the sclerosis has been produced by the long-continued strain on the arterial walls caused by the hypertension. As a rule, however, cases of hypertension do not live long enough to develop a high degree of arteriosclerosis. It is clear that some underlying factor not accessible to our present methods of examination must be responsible for the occurrence of high blood pressure in arteriosclerosis. This factor we may assume on general grounds to lie in some disturbance of the splanchnic circulation. In health a certain functional interchange or "give and take" exists between the systemic and splanchnic circuits, the systemic drawing upon the splanchnic at need and at other times using it as a storage for blood.

In disease this "normal balance" may be greatly disturbed and if the splanchnic vessels be the seat of sclerosis their reserve capacity will be reduced and the systemic arterial pressure in consequence raised and maintained above normal. This is practically the conclusion of Hasenfeld and Hirsch, who from clinical and pathological data contend that it is only when the vessels of the splanchnic area or the aorta above the diaphragm are diseased that high pressure develops in arteriosclerosis. For the present at least we may assume that when arterial pressure is persistently raised the terminal divisions of the vascular system (splanchnic and systemic) are principally involved in the sclerosis. This is of the highest practical importance for arteriosclerosis of the splanchnics and arterioles cannot be recognized by physical investigation during life, but may be inferred to exist with a fair degree of certainty from blood pressure observations. The prognosis in this type of case is very different and far graver than in that other order of sclerotics with stiff chalky arteries, but no tension. As Cook has emphasized, the outlook in the case of a robust looking man of fifty-five with a blood pressure of 200 mm., even with no appreciable arterial degeneration, is not so good as in the case of a man of sixty-five with rigid arteries and a pressure of 130 mm. We find many examples of the latter type in every old

peoples' home where they live on year after year eventually dying of an atrophy, cerebral or cardiac. The patient with hypertension is in daily danger of apoplexy or heart failure. The element of danger is the tension.

Fraenkel and Hasenfeld have pointed out that corpulent persons of a sedentary habit are prone to develop sclerosis of the splanchnic vessels with high blood pressure. There is much reason to believe that such cases are toxic in origin and it is probable that the chief source of the pressor toxins is the digestive organs. The argument of clinical experience lends weight to the contention of Russel that in non-nephritic cases the hypertension is caused by the presence in the blood of substances which are absorbed from the alimentary tract and are the product in one form or another of what has been swallowed as necessary food or as unnecessary indulgence. This does not imply that the big feeder must necessarily develop splanchnic sclerosis and the small feeder escape. The essental factor will prove to be the digestive and eliminative competency of the individual and the relative suitability of his diet. In summing up the relation between arteriosclerosis and high blood pressure we must acknowledge in the first place that the thickening of the vessel walls incident to age is capable of causing a gradual, but by no means extreme rise in the average arterial pressure. We see this in the slow increase of average pressure readings as life advances, a pressure of 140 mm. being accounted normal for a man of sixty-five years, whereas his son will have a pressure of 120 mm. and his grandson 100 mm. Should the pressure of such a man register persistently above 160 mm. and with this his heart show enlargement he may be regarded as having hypertension and we must invoke some cause other than his arteriosclerosis to explain it. Such a development means that in addition to a thickened artery he has a constricted one, the former an anatomical change, the latter a spastic condition produced by some toxic excitant circulating in the blood and causing hypertonic contraction of the arterioles, splanchnic or systemic (Russel).

It is hardly necessary to urge the importance of carefully investigating the condition of the urine in every instance of hypertension. The frequency with which chronic renal disease is associated with cardiovascular changes is well known and no factor is so potent as nephritis in the production of high blood pressure. If a diagnosis of chronic interstitial nephritis can be made it is not necessary to search farther for the cause of high pressure. At the same time it is to be remembered that greater care than ordinary is required to diagnose nephritis in the presence of arteriosclerosis with high blood pressure, owing to the fact that some degree of atrophy of the kidneys, manifested by slight albuminuria and casts, is almost always present as a consequence of those organs sharing in the general vascular deterioration. Moreover, almost all cases, no matter of what origin, showing blood pressures over 200 mm. will display some albumin in

the urine. It is unfortunate that no clear distinction is made in clinical literature between arteriosclerotic renal atrophy, and true contracting kidney. Every experienced clinician knows how vast the difference is in course, prognosis, and treatment between the two conditions, and yet it is only from experience and not from medical literature that one learns to appreciate the distinction. Chronic interstitial nephritis is an inveterate organic lesion showing severe toxic manifestations, a steady and even rapid downward progression, is not amenable to treatment, and has a bad prognosis. In every respect, no matter how similar may be its urinary and physical indications, arteriosclerotic renal atrophy is the opposite of this, being slow in development and progress as is the case with the sclerotic atrophies generally. A distinction between the two is important owing to the difference in prognosis.

Chronic interstitial nephritis gives rise to the highest systolic readings that are observed clinically, pressures of 300 mm. and higher being recorded. The highest record I have is 285 mm. in a young woman, who died a fortnight after in uremic coma. In a study of 60 cases of chronic nephritis[1] the average pressure was 190 mm. Chronic nephritis is essentially a disease of systemic scope, involving the heart and arteries as well as the kidneys. Arterial hypertension is one of its salient features. Notwithstanding occasional exceptions to the rule, high pressure is so significant that it constitutes one of the most valuable diagnostic indications of that disease. The use of the sphygmomanometer and the discovery of high pressure will at once put the observer on the alert.

The attempt to establish a working hypothesis to account for all the varieties of hypertension leads us face to face with the toxic theory. Arterial hypertension is best exemplified as it occurs in a group of chronic diseases having as their common essential characteristic, toxemia. In all probability the *materies morbi* consists of certain abnormal biochemical products present in the circulating blood. This is apparent in scarlatinal nephritis in which the tension rises a few hours after the appearance of albumin in the urine, entirely too early for the rise to be explained by the formation of arterial fibrosis. The high tension of uremia is another instance in point. For laboratory proofs we have the well-known fact that the pressor principle of adrenal and pituitary glands and also certain drugs (ergot, nicotin, digitalis) will raise blood pressure. That hypertension is a functional effect, as well as an organic product, we may infer from the clinical observation that measures designed to detoxicate the system (diet, sweats, cathartics) will result in some reduction of pressure in most cases of hypertension, and the therapeutic action of the nitrites could not be secured did the condition rest solely on a basis of structural change.

[1] Jour. Amer. Med. Assoc., April 13, 1907.

The end-effects of long continued high blood pressure are manifested principally in the heart and arteries. The arterial walls reacting to the excessive mechanical strain undergo a progressive structural deterioration to the great prejudice of their normal histology and vasomotor tonus. In the end vasomotor response may be so seriously disturbed that nitrites may fail to lower blood pressure. Peripheral resistence increases in this manner with the stage of the disease. A somewhat parallel sequence of events is apparent in the heart. At first in response to overwork the myocardium hypertrophies just as does the myarterium. The peripheral retard being persistent and increasing and the heart reserve limited, myocardial insufficiency becomes inevitable. The heart at first hypertrophies and then dilates in the face of continued overstrain.

The complications of arterial hypertension will be determined by the ability of the different organs to withstand the strain. On the part of the heart we observe hyposystole and asystole, of the arteries atheroma, of the kidneys albuminuria and uremia. A cerebral vessel may give way and apoplexy close the scene.

The symptomatology of arterial hypertension is general rather than special. There may be an entire lack of symptoms until vertigo or an attack of acute dyspnœa alarms the patient, or some cerebral accident occurs. The condition is often revealed quite unexpectedly during examination for life insurance. Frequently the earliest symptoms are of nervous type, irritability, depression of spirits, disturbed sleep, or it may be that the patient complains of bilious symptoms, flatulency, constipation, headaches, and vertigo. There is usually precordial discomfort and dyspnœa following effort and the patient rises once or more at night time to void urine. As a rule the night urine exceeds in quantity that passed during the day. A symptom noted in a number of my cases is a severe paroxysmal flatulency nocturnal in occurrence or developing on exertion. During the intervals between attacks there may be no complaint of flatulency, and examination at any time may fail to reveal special tympany or distention. Shortly after retiring for the night the patient may experience a feeling of distention and oppression across the lower chest or he may awake after a period of sound sleep with a feeling as if the stomach were full of gas. Instinctively he strives to relieve himself by gulping, drinking hot aromatic or alcoholic drinks, and the fact that comfort is reëstablished, frequently after an hour or more, only when he has belched freely, confirms him in his idea that it is indigestion. The breathing is usually hurried, and palpitation may coincide. Attacks of this nature may develop after exertion, especially soon after meals and most frequently during the early part of the day. Bending over, lifting, straining at stool may precipitate the symptom. This development is probably similar in character and significance to cardiac asthma and denotes insufficiency of the right heart. All of the patients with this symptom whom I have seen,

have had enlarged livers and one a well marked jugular pulse. I have come to regard flatulency of irregular and paroxysmal occurrence in mature individuals as extremely significant, and I believe that every such case should be carefully investigated as to the cardiovascular condition. A typical instance of this character is the following case.

S. F., aged seventy-six years, and weighing one-hundred and ninety pounds, is a retired merchant of means, and boasts that he has never been sick in his life. He confesses that he has been a very hearty eater and heavy smoker, and has for years been constipated. Although rather spare in his limbs he is round-bellied and his face is ruddy and somewhat pigmented. He complains that for some months he has been greatly troubled with attacks of explosive belching of gas developing during exertion and interfering greatly with his activities. These attacks are frequent during the early part of the day, especially after breakfast and occur comparatively seldom and less severely toward evening. They come on during walking, especially if the weather is cold or it is windy or the walking rough. He is compelled to sit down or lean against a fence or building until he had relieved himself by belching. There is little intestinal flatus, and but slight dyspnœa between the attacks. He is somewhat puffy on exertion. He rises two to four times at night to void urine. He is found on examination to have a greatly enlarged heart, the apex 17 cm. from the midsternum; and a systolic blow at the mitral area reveals the lea of dilatation. The blood pressure ranges from 190 mm. to 225 mm. There are no urinary indications of nephritis. This patient remained under observation for two months with slight benefit to his symptoms and then departed south to a more agreeable winter climate. Two weeks after his departure he died of acute heart failure.

It is noted frequently by patients with hypertension that smoking causes restlessness and cerebral discomfort and that heavy meals are not so well borne as formerly. Women are apt to complain of flushing and burning of the face, and if in the middle period of life are apt to ascribe it to the climacteric. I have noted as a prominent symptom in two cases pains of an anginoid character referred to the precordium, the left arm, or abdomen following exertion. Tachycardia and palpitation are complained of. As a sign of great significance and rather grave import, as it indicates the beginning of hyposystole, is dyspnœa on lying down. The explanation of this development lies in the fact that the arterioles being contracted the blood collects unduly in the veins especially the splanchnic veins. When the patient is upright these veins act as a reservoir, but when he lies down the force of gravity tends to empty them into the right heart. This leads to overdistention of the pulmonary capillaries and dyspnœa. The only way rightly to interpret these symptoms is to take the blood pressure. With every individual of middle age com-

plaining of persistent functional disturbance this precaution should never be omitted.

Examination of the heart in cases of hypertension will reveal enlargement of that organ. This may sometimes be difficult of detection in fat individuals and in women with pendant breasts. In the earlier stages the heart sounds are usually clear, the first tone booming and prolonged, the aortic second loud, valvular, and ringing. If the ventricle has dilated the murmur of relative mitral insufficiency may be heard. Late in the case with the heart badly disorganized, the patient dropsical, and the pulse small and arrhythmic, it may become extremely difficult to determine whether the case is one of cardiac failure from prolonged hypertension or a valve lesion in the final stage of incompensation. The sphygmomanometer may afford us no help at this juncture, owing to the failure of the ventricle having so impaired the support of the circulation as to induce secondary low blood pressure. One must then fall back upon the history and the general features of the case to decide the point.

Analysis of the urine in arterial hypertension may reveal no morbid elements from the kidney, although in cases of some standing a few hyaline casts will usually be found. If chronic nephritis exists the usual urinary characteristics of that disease will appear to point to the origin of the hypertension. Renal permeability to albumin seems to be overcome when the blood pressure reaches or exceeds 200 mm., so that cases with very high pressures, whether primarily nephritis or not, usually have albumin in the urine.

THE USE AND ABUSE OF GASTRO-ENTEROSTOMY.[1]

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Gastro-enterostomy, "the keystone of gastric surgery," was first performed in 1881. Today this operation is being frequently made. The object of this paper is to excite discussion upon the bearing of the operation along the lines where it is proper as well as improper. The risks of gastro-enterostomy performed by an experienced surgeon are inconsiderable. The mortality of gastro-enterostomy in benign disease is low, 1 to 2.5 per cent. This is fortunate in a sense, and yet unfortunate if it encourages the performance of the operation in conditions in which it is not properly indicated.

A most important, essential, and interesting point is that metab-

[1] Read at a meeting of the Manhattan Medical Society, New York, December 17, 1909.

olism after gastro-enterostomy is not interfered with to the degree of making the operation objectionable on this account. It has been clearly demonstrated by observers, particularlly Paterson, that metabolism is in no way seriously altered. It has been my experience, after observing a large number of cases several years after operation, that not only has the patient's digestive ability been in no way impaired, but that he was able to take more freely of food, even such as could not be digested before. The ultimate results of gastro-enterostomy have been most satisfactory. This has been demonstrated by collected cases, notably those of Mayo, Moynihan, myself, and others.

Formerly the most dreaded complication of gastro-enterostomy was regurgitant vomiting. This, since the no-loop operation has been done, is practically a thing of the past. I was unfortunate enough to see a few of these cases when I practised the long-loop operation, but have not had a case of the kind since doing the no-loop operation. One of my cases of regurgitant vomiting necessitated five operations before I was able to correct it. The cause of regurgitant vomiting was believed to be the presence of bile in the stomach from the afferent loop, but it has been established by experiments on dogs that bile in the stomach has no injurious effect on digestion or the general health. In confirmation of this, Moynihan has reported a case in which the result of rupture of the intestine at the junction of the duodenum and jejunum necessitated closing the duodenal end of the bowel and transplanting the jejunal end into the stomach, thereby causing all the bile to enter the stomach through the pylorus; the patient never suffered from vomiting, and remained in good health several weeks after the accident, until his death, which was caused by perforative peritonitis, due to the Murphy button. Some surgeons, notably Kehr, perform cholecystogastrostomy in preference to cholecystocolostomy.

It has been my practice to place an anchor suture one-half to one inch distant from the commencement of the efferent portion of the bowel, in this wise preventing angulation and consequent spur formation, thus minimizing the chances of obstruction to the onward passage of the contents of the afferent loop. I believe that the cause of regurgitant vomiting probably has been a mechanical defect at the site of the anastomotic opening, therefore faulty technique. Other complications after the operation, as detailed by Moynihan, are hemorrhage, internal hernia, separation of united viscera (leakage), formation of adhesions at or near the point of new opening, peptic ulcer, pneumonia, and diarrhœa.

I have never encountered hemorrhage. I have never had a case of internal hernia or separation of united viscera, or leakage. Internal hernia is prevented by careful suture of the margin of the opening in the mesocolon to the wall of the stomach. To my mind that is more rational than suture of the margin of the opening in the

mesocolon to the bowel. Suture of the mesocolon to the bowel, if followed by contraction of the marginal mesocolon, may so constrict the bowel as to cause obstruction of the anastomotic opening and interfere with the passage of the contents of the duodenum into the bowel beyond the anastomotic opening. In this connection I might say that the surgeon should be a good cutter and a good sewer to avoid mechanical complications. I have seen the formation of adhesions at or near the new opening; and also pneumonia; but never peptic ulcer or profuse diarrhœa.

The conditions for which the operation of gastro-enterostomy is indicated are: Chronic gastric and duodenal ulcer, with their sequels, perforation, recurrent hemorrhage, and cicatricial contraction; carcinoma of the pylorus, in connection with excision or alone by way of palliative treatment; benign pyloric obstruction resulting from stricture, adhesions, or angulation; gastric tetany; gastroptosis, with loss of stomach motility, and therefore with stagnation and usually more or less dilatation; chronic dilatation, without gastroptosis, with stagnation from loss of motility; infantile hypertrophic stenosis of the pylorus; duodenal cancer or tumor causing obstruction; duodenal fistula; the rare cases of plastic linitis of the stomach, in which the hypertrophy of the walls reduces the stomach to such a size that only liquid in small quantities may be taken and retained.

The conditions in which the operation is contra-indicated, therefore, in which the operation is an abuse, are: Acute dilatation of the stomach, gastric neuroses, dilatation without stagnation, advanced carcinoma of the pylorus, and gastric crises.

This operation does good in chronic gastric and duodenal ulcer by diminishing the acidity, by abating pylorospasm, and possibly by allowing the entrance of greater quantities of bile into the stomach, removing the condition which has prevented healing of the ulcer. About 80 per cent. of patients operated on for gastric ulcer by gastro-enterostomy recover. That gastric ulcer is frequently multiple must be acknowledged. It is true that we have no pathological evidence that gastric ulcer is healed following the operation of gastro-enterostomy, yet we have clear clinical evidence that this is so. The mortality of gastric ulcer treated medically is about 20 per cent., and at least 50 per cent. of cures, so-called, relapse, and probably not 25 per cent. of patients treated medically are really cured. The proportion of cases of relapse after cure following gastro-enterostomy is about 10 per cent.

If the ulcer is not located at the pylorus, the latter therefore opens, and the contents of the stomach will partly pass through it as well as the new opening. The churning and propulsive movements of the stomach, which are later taken up by the pylorus and carried on through the duodenum and small intestine, are not interfered with. Some of the stomach contents pass through the new opening, as the

cut circular muscular fibers, the agents in the propulsive movements, are attached to the margins of the new opening, and in contracting separate the margins of the opening and thus allow the stomach partly to empty through this route. In complete pyloric obstruction all of the gastric contents, as a matter of course, pass out by way of the anastomotic opening.

As this paper deals with gastro-enterostomy alone, I have said nothing about the excision of the gastric ulcer. There is a great difference of opinion as to the relative merits of excision and gastro-enterostomy. The strongest argument in favor of excision is the likelihood of carcinoma becoming engrafted on the ulcer scar. The decision, pro or con, is best made at the time of operation, according to the appearance and consistency of the ulcer. A thickened, greatly indurated ulcer is better excised, since it is impossible to determine whether malignant changes may not already have taken place, and experience tells us that a considerable percentage of those excessively hard and thickened ulcers do show carcinomatous changes. Unless there is a suspicion of malignancy, however, gastro-enterostomy is the operation of choice, as showing a considerably smaller mortality.

The operation is strongly indicated in cases of recurrent bleeding, in which the intervals between bleedings are growing shorter, and the amount of blood lost the equivalent or more than on previous occasions. The following case is an illustration of operation for this condition:

Miss ——, aged twenty-six years. In 1903 she noticed the first symptoms, which subsequently suggested gastric ulcer. She was miserable for two years, when she was again attacked with symptoms referable to the stomach. In August, 1907, she had an attack of severe abdominal pain, continuing for three days. Nothing remained in her stomach; she suffered from nausea and vomiting, which continued for two weeks. On August 23, after taking a small quantity of beef juice, she had a very severe hemorrhage, followed by several smaller hemorrhages at intervals. I saw the patient on August 30, with the physician in charge, Dr. Branson. As she had not vomited for two or three days before my visit, and her condition was so wretched, we agreed to defer operation for a few days.

Operation, September 3, 1907. Exposure of the stomach showed a saddleback ulcer on the lesser curvature, four inches from the pylorus, with greatly indurated edges. Subtotal gastrectomy and gastro-enterostomy were done. Recovery was uneventful.

In hour-glass stomach gastrojejunostomy makes a part of the necessary interference. This operation alone will seldom suffice, as when made in the pyloric pouch only the obstruction to the passage of food from the cardiac pouch still exists, and when made in the cardiac pouch alone it will not drain the pyloric pouch; hence

it is necessary, in addition to gastro-enterostomy at the pyloric pouch, to do a gastrogastrostomy or gastroplasty, so as to place the two pouches in communication.

Gastro-enterostomy is indicated when a perforated ulcer of the stomach or duodenum is sutured, if the patient is bearing the anesthetic well. It has always been my practice to do this operation in connection with the closure of the ulcer. I know there are many surgeons who take the opposte stand; nevertheless, I am of the opinion that it is proper to do it if the patients are operated on comparatively early. Patients that are operated on late after perforation die, do what one will. The additional time which gastro-enterostomy takes when closing a perforated ulcer of the duodenum or stomach is a matter of no moment if the operation is done at a timely season. The chief advantages of making the anastomosis is to make the patient permanently well after he recovers from the closure of the perforation, which is too frequently not the case when this is not done, the patient continuing to suffer from indigestion. In the event that another ulcer has been overlooked, this places the patient in the best position for permanent relief. The surgeon will have more confidence in closing the ulcer, particularly if it has indurated borders, and he will not fear having caused too much obstruction to the lumen of the viscus. The operation of gastro-enterostomy puts the part at rest and makes healing certain and quicker, and therefore lessens the risk of leakage; allows us to feed our patients earlier, which is of some moment in a certain percentage of cases. Yet I may say here that I never had any trouble in nourishing my patients for the first two or three days by the bowel, by giving saline solutions and expressed beef juice. In fact, I think patients, as a rule, do not require anything in the shape of food for two or three days after the operation.

In carcinoma involving the pyloric end of the stomach, too far advanced for radical operation, and the patient's general condition being fairly good, and indicating that, if able to take nourishment, his life would be prolonged for several months and his comfort increased, the operation is warrantable. I believe that gastro-enterostomy is often performed in carcinoma of the stomach that is radically inoperable, when it had better not be done, as it only adds misery to misery.

That gastro-enterostomy is the only alternative in benign pyloric obstruction due to cicatricial contraction, adhesions, or angulation goes without saying. The exception would be an occasional Finney operation in exudative contraction, yet I am of the opinion that gastro-enterostomy here is the better operation from the standpoint of ultimate results.

In gastroptosis with or without dilatation and with stagnation, and in dilatation with stagnation, gastro-enterostomy is strongly indicated, providing the patient has received treatment in the shape

of lavage, diet, gymnastics, and attention to general hygiene, without recovery. In dilatation, unless the case yields very quickly to diet and treatment, it should be explored. It is not fair to the patient to withhold relief in the presence of chronic dyspepsia that does not yield to medical means, thus exposing the patient to the greater risks of delay. The frequency with which chronic dyspepsia proves at operation to be due to some tangible cause is a striking fact in practice. The necessity for the habitual use of the stomach tube is sufficient indication for gastro-enterostomy.

In infantile hypertrophic stenosis, in which the symptoms persist in spite of lavage and careful feeding, gastro-enterostomy promises most, but must not be deferred until hope of cure is out of the question. In duodenal ulcer the rationale for gastro-enterostomy is the same as in gastric ulcer. In duodenal tumor, duodenal fistula, and gastric tetany, it may be necessary to resort to this operation. In plastic linitis I have seen excellent results. At present I have in mind one case of a doctor who had not been able to take anything but liquids, and these in small quantities, for a number of years, owing to this condtiion. Following gastro-enterostomy he was restored to practically a normal condition. The stomach in this patient was one and one-half inches in vertical diameter, two inches in fore and aft, with walls an inch thick.

The operation is abused if done in advanced cases of carcinoma with marked cachexia. I am quite sure that in many cases of carcinoma of the pylorus the operation is ill advised. The mortality of subtotal gastrectomy in the latter class of cases is so little greater than gastro-enterostomy, that I question the propriety of gastro-enterostomy in the presence of a growth that can be excised without injuring the pancreas, if the glandular involvement be not too extensive. Injury of the pancreas, if followed by escape of the pancreatic ferments, which cause necrosis of the tissues with which they come in contact, is a serious condition. When the profession awakes to the importance of opening the abdomen early in the case of chronic dyspeptics, gastro-enterostomy will have a small place in the surgery of carcinoma of the stomach, except in connection with excision. That the operation is much abused if done in cases of gastric crises we will all agree.

In acute dilatation of the stomach the operation of gastro-enterostomy will never be required if the stomach tube is used earlier and oftener in persistent nausea, not waiting until there is vomiting in that class of cases in which we are not surprised to see it. In my surgical work the stomach tube is used to the exclusion of any and all medication, formerly and still believed by many to be worth a trial. The time lost in giving medicines, with the hope that they will do good, is the very time that lavage is to be practised if we are to prevent this serious complication. In chronic dilatation, with or without prolapse, if there be no motor insufficiency or stagnation, the operation is useless.

There is no doubt that there are certain morbid gastric conditions which have been, and are still, classified as neuroses. What concerns us particularly is that set of gastric symptoms classed grossly as "nervous dyspepsia." Under this general term have been grouped the most diverse symptom-complexes, with, as a rule, but little understanding of the underlying principles of the case. It is true that there are certain disturbances in the gastric function, motor, secretory, and sensory, for which we can, by the minutest examination, find no organic basis. Besides grouping them into these three classes, we may also classify them as conditions of irritation or depression. Thus, gastralgia, nausea, and gastric hyperesthesia are prominent types of sensory disturbances; hyperchlorhydria and hypersecretion are well-known types of secretory disturbances; while atony, pylorospasm, and pyloric insufficiency represent well-known varieties of motor disturbance. Needless to say, motor, sensory, and secretory aberrations may all be combined in a given case, and it is by various combinations that the different types of so-called "nervous dyspepsia" are produced.

As a fundamental principle, we can safely state that a gastric neurosis without other neuroses or neurasthenic conditions is a most rare thing. The gastric symptoms, however, may so overshadow all others that attention is directed only to them.

In sensory disturbances we find more or less anorexia, or at least capriciousness of appetite, in almost every case. It is such a constant symptom that it is of little value; practically every sufferer from every form of gastric disease, real or imagined, complains of it at one time or another. True gastralgia I have found rarely. Of the secretory disturbances, hyperchlorhydria is the most important. Our ability to diagnosticate the condition by analyses of stomach contents and secretions is not great, yet extreme cases can be diagnosticated in this way, and do at times occur in the absence of anything that would seem to account for the condition. Atony of the stomach also cannot at times be considered as anything but a neurosis, and its treatment falls fully as much within the province of the surgeon as of the internist. In the diagnosis we are again confronted by the lack of exactness of methods of examination and the difficulty of fixing a standard with wide enough limitations to include all normal cases, and yet of sufficient definiteness to be a standard.

Finally, we have that vague group of symptoms, sensory, motor, and secretory combined, which, in the absence of any definite or tangible demarcation, has been called "nervous dyspepsia." It includes definite feelings of distress, pain or heaviness in the epigastric region, eructations, anorexia, gastric torpor rather than marked atony, intervals of excess of acid secretion, and an associated intestinal derangement, with almost invariable constipation.

The most important features in the diagnosis of any gastric

neurosis is the eliciting of a careful history, which will show the general neurasthenic condition of the patient. The presence of a manifest general nervous breakdown with an undoubted neurasthenia would at once predispose us to consider any gastric symptoms present as but local signs of a general process. Again, this run-down condition may be a secondary neurasthenia, due to a primary lesion which underlies both it, indirectly, and the primary condition of the stomach most directly. A patient with a latent but not symptomless gastric ulcer would soon show gastric symptoms, which might be considered nervous in origin, as well as a general neurasthenic condition, due to his sufferings.

Carcinoma in its early stages is much more often considered as a gastric catarrh or nervous dyspepsia than it is recognized. Anorexia, followed by the symptoms of a vague chronic gastritis or neurosis, when it occurs in a middle-aged person, is a condition which should excite our greatest apprehension, and be dismissed from consideration only after the most careful examination has been made—after the case, if obstinate, has come to operation.

Punctate ulceration of the stomach mucosa with small, early bleeding points may involve almost, if not quite, all of the gastric mucosa. In the absence of the classical signs of ulcer, which we often have in this condition, the hyperchlorhydria present has often been mistaken for the main lesion.

It has always been my opinion that in very many of the cases of vomiting regarded as primary neuroses we have really a symptom only of some lesion in or outside of the stomach which, for some reason or other, we have been unable to determine. Vomiting as a pure motor neurosis is regarded nowadays as far less frequent than it was thirty years ago, yet we occasionally see it.

The surgeon's principal duty as regards the true neuroses of the stomach is to recognize them, to separate them from secondary dyspeptic conditions due to lesions which perhaps it is within his province to treat. I regard the proposition to operate on these cases for the mental effect upon their general neurasthenic or hysterical condition as unsafe, illogical, and as setting a most dangerous precedent.

There is no exception, perhaps, to the general statement that gastric neuroses per se are not within the province of the surgeon. If we consider gastric atony and ptosis as really neuroses, when they are apparently primary, they form the exception. There are certain of these cases in which all medical and general treatment is unavailing, while a gastro-enterostomy promptly leads to recovery, by furnishing the stomach with drainage, which by its own force it is unable to secure. In ptosis I believe that gastro-enterostomy is the only logical procedure. This is true even in some instances in which the pylorus is entirely patulous. While I believe that an

occlusion of the pylorus is the main indication for gastro-enterostomy, I do not think it is the only one.

To make a gastro-enterostomy upon a patient with gastric neurosis pure and simple is nothing short of a catastrophe. Within the past year a patient came under my observation who had had several operations performed, the last of which was a gastro-enterostomy. The patient, a typical neurasthenic, as a matter of course was not only not benefited by the latter procedure, but made very much worse. After having her under my care for a number of days I determined to restore her stomach and intestines to a normal condition, minus the amount of bowel necessary for her to lose in order to cut out the portion involved in the anastomosis; this I did, with closure of the stomach, the patient being markedly benefited thereby for a time. I have recently learned that she is vomiting again and her condition is practically the same as before the first operation. There could be no better example of the futility of operation in gastric neuroses.

Being jealous of the benefits that surgery has conferred upon humanity, not the least of which are in this field, I do not wish to have discredit cast upon her efforts by operations performed upon improper indications. It is with some trepidation, however, that I advise against operation in gastric neuroses, simply because so many cases are thus incorrectly diagnosticated which would afford brilliant surgical cures. In giving this advice, therefore, it is with a plea for more careful observation to rule out any possibility of an organic lesion being accountable for the symptoms. We are justified in considering only those cases as neuroses which give a history clearly indicating other neurotic stigmas, with symptoms that vary greatly without apparent cause, or as the result of emotional states, and which give to careful observation no clue to an organic lesion. Better that such cases should come to exploration occasionally than to miss many true surgical cases. But I do not concur in the advice to operate upon these cases knowingly, nor, having unwittingly explored the stomach in such a case, to make a gastro-enterostomy or any other operation in the absence of a definite physical indication.

In conclusion, it is fitting for me to say that every surgeon should first be a physician. The surgeon should understand disease, its physical signs, and its differential diagnosis. The surgeon who does not possess this knowledge is not in a position to advise treatment. The surgeon should not be the mere human tool of the physician. I regret to say that surgeons are of two classes—the surgeon and the operator; the combination is what makes the true surgeon. It is to be regretted that the laity too often regard the surgeon as the last man to be called in. How often their distaste for the surgeon has been the cause of the fatality!

HAVE WE MADE ANY PROGRESS IN THE TREATMENT OF GONORRHŒA?[1]

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So much has been said and written on the theme of gonorrhœa and its treatment that you may be disposed to ask why I should have chosen it for this evening's discussion. The answer to this question is, that for an indefinite time the impression has been growing into a conviction that we are getting better results than formerly in the treatment of gonorrhœa. Not that we are shortening the duration of an attack; for, although in some cases the disease can be promptly throttled, its duration still averages from four to six weeks; but (1) we are now able to mitigate the sufferings of the first or acute period, say of the first week. (2) We believe that there are fewer complications, and a diminished liability to them; as, for instance, to posterior urethritis with its liability to inflammation of the contiguous structures. (3) There is less tendency to become chronic and to the development of that formerly frequent sequel of gonorrhœa, stricture of the urethra. (4) It is now easier to insure the patient's attention to treatment, for there is a wider and better understanding of the danger of infection by latent gonorrhœa.

You will admit that anything relating to this disease continues to be of great importance; for apparently there is no lessening in the number of cases, but, on the contrary, a gradual and steady increase in their number. Dr. Victor C. Pedersen, who is in charge of the Hudson Street House of Relief on the west side of the city, tells me that there is a "normal" increase in the number of the cases of gonorrhœa. At this institution there are from 7800 to 8000 new patients per annum, and from 60 per cent. to 70 per cent. of these are cases of acute gonorrhœa. Dr. Swinburne, on duty at the Good Samaritan Hospital, on the east side of the city, informs me that in this hospital there are from 50 per cent. to 60 per cent. of new cases of acute gonorrhœa per annum. It is evident that as yet no propaganda of scientific instruction has reached this stratum of society, and, according to Morrow, Julienne, and others, the general morbidity in women, men, and children as a result of gonorrhœa is so great as to warrant the term *alarming;* and at all events it is sufficient to arouse within us the wish to do what we can to lessen the grave scourge.

Morrow says: "It is a conservative estimate that fully one-eighth of all human disease and suffering comes from this source. Moreover, the incidence of these diseases falls most heavily upon

[1] An address delivered at a meeting of the Society of the Alumni of Bellevue Hospital, New York, March 3, 1909.

the young during the most active and productive period of life. It is a fact worthy of consideration that every year in this country 770,000 males reach the age of early maturity; that is, they approach the danger zone of initial debauch. It may be affirmed that under existing conditions at least 60 per cent., or over 450,000, of these young men will some time during life become infected with venereal disease, if the experience of the past is to be expected as a criterion of the future. Twenty per cent. of these infections will occur before their twenty-first year, 50 per cent. before their twenty-fifth year, and more than 80 per cent. before they pass their thirtieth year. These 450,000 infections, be it understood, represent the venereal morbidity incident to the male product in a single year. Each succeeding group of males who pass the sixteenth year furnishes its quota of victims, so that the total morbidity from this constantly accumulative growth forms an immense aggregate. . . . There is abundant statistical evidence to show that 80 per cent. of the deaths from inflammatory diseases peculiar to women, 75 per cent. of all special surgical operations performed on women, and over 60 per cent. of all the work done by specialists in diseases of women are the result of specific infection. In addition, 50 per cent. or more of these infected women are rendered absolutely and irremediably sterile, and many are condemned to life-long invalidism. From 70 to 80 per cent. of the ophthalmia which blots out the eyes of babies, and 15 to 25 per cent. of all blindness is caused by the gonococcus infection."

On the other hand, Dr. E. A. Davis and Dr. Gehring[2] claim that the number of cases of ophthalmia neonatorum in the dispensaries is diminishing, because of better understanding of the care of the baby at birth.

Now the question naturally arises, are these impressions as to improvement in treatment shared by other observers? And will the opinions and teachings of others in the practice of genito-urinary surgery deepen or efface these impressions? Let us see what answer we shall get from authorities. I will quote freely from the literature of the subject.

1. What has been accomplished in relation to the duration of the disease? In my own practice, although admitting that the majority of cases last from four to six weeks, there are others (more in number than formerly), especially in the higher social strata, which terminate in two or three weeks. But there is an interesting unanimity of opinion on the part of authorities that the disease takes from four to six weeks to run its course in those cases, which do not become chronic.

For example, Watson and Cunningham[3] define "cure" as follows:

[2] Oral communication.

[3] Diseases and Surgery of the Genito-urinary System, 1909.

"By cure we mean not necessarily the cessation of the discharge, but its non-recurrence upon omitting the treatment and upon resuming an ordinary manner of living." They then say: "One frequently hears the claim that gonorrhœa can be cured within a week or ten days, or at least a fortnight. Personally we have no sense of shame in frankly confessing our inability to accomplish such results, *as a rule,* although we sometimes succeed in so doing. We consider ourselves and the patient fortunate if we obtain a cure at any time less than six weeks." R. W. Taylor[4] states: "In favorable cases a cure may be brought about in four to six weeks. Occasionally some patients get well in three or four weeks." White and Martin[5] state that the "prognosis, under favorable conditions, is good for recovery by the eighth week;" while Greene and Brooks,[6] without making a definite statement as to time, think that "it is better to postpone the active local treatment of urethritis until after the acute stage is passed and the discharge first becomes mucopurulent. This is generally about the fourth to the sixth week after the onset of the disease." Hyde and Montgomery are even less sanguine and consider that "usually a first attack, with favorable circumstances and good treatment, recovers in from five to eight weeks." Morton also states that "the percentage of recoveries in two or three weeks is small, and that the usual duration is six weeks." Finger and Casper, representative of the Germans, are unqualifiedly pessimistic, and while admitting that the disease ends in from five to six weeks, state that the prognosis is doubtful. On the other hand, Keyes, Senior and Junior[7] are decidedly optimistic, and quite positively state the duration to be "untreated, six weeks or more; but curable within two or three weeks by the irrigation method."

2. In regard to the mitigation of symptoms, although this amelioration may be inferred from such statements as "in from four to ten days all obvious discharge ceases," etc., I find very few definite statements; but as a result of my experience it may be confidently said that prompt treatment by one of the albuminoid preparations of silver (organic compounds), together with judicious hygienic measures resolutely carried out, will reduce the activity of the infection, proportionately subdue the inflammatory symptoms, and possibly modify the whole course of the attack.

3. In regard to the complications of gonorrhœa, we are met by contradictory statements and confusion of opinions. A study of systematic writers, in collaboration with Dr. Edward Preble, shows that the percentage of frequency of complications varies so much with individual experience that it is impossible to prove by the

[4] Genito-urinary and Venereal Diseases, 1900.
[5] Genito-urinary Diseases, 1907.
[6] Diseases of the Genito-urinary Organs and the Kidneys, 1908.
[7] Surgical Diseases of the Genito-urinary Organs, 1903, pp. 119 to 124.

evidence that complications were any more prevalent under old methods than at the present time. So far as blood infections and remote metastases are concerned, there are virtually no statistics given, while for epididymitis, cystitis, prostatitis, spermocystitis, etc., the figures show every variation. In Wossidlow's monograph (1903) the conclusions agree with those of other writers; namely, that acute posterior urethritis, according to the majority, is an all but universal sequel, while other writers find it much less frequent. Statistics of acute prostatitis vary from 3 to 70 per cent.; spermatocystitis is said by some to be very rare, while others make it extremely common. There seem to be no figures for cystitis, as it is too readily confounded with posterior urethritis and prostatitis.

Uhle and McKinney[8] cite the combined statistics of Rollet, Tarnowsky, Jullien, and Finger—11,972 cases of gonorrhœa, with 2244 cases of epididymitis, or 18.7 per cent. The authors' own material represented 16 per cent. Neuberger[9] quotes Jordan, of Moscow, who compiled statistics which show that 30 per cent. of gonorrhœal patients suffer from epididymitis in hospital practice. In dispensaries and private practice the proportion, according to Jordan, varies from 7 to 17.3 per cent. The figures for dispensary patients are 11.7, but if the history of the cases was taken into account the proportion increased to 27.8 per cent. The joint testimony of several authors is to the effect that from 80 to 90 per cent. of gonorrhœal patients develop posterior urethritis. The author gives a series of 200 cases treated in the early period with protargol injections followed by Janet irrigations when the subacute stage was nearly over, with but six cases of epididymitis, or 3 per cent. Neisser does not use irrigations in the acute period, relying upon injections of the prolonged type. His proportion of epididymitis was 9 per cent. Tauska[10] gives an analysis of 17 statistics, making the percentage vary from 3.2 to 29.2 per cent., the average being 15 per cent. His material was 674 cases of gonorrhœa, 75, or 11.1 per cent., having epididymitis on admission, while 18 cases gave a history of the complication. The total of 93 cases was 13.8 per cent.

Lewin and Bohn[11] present a series of personal statistics on acute spermatocystitis; the article also gives incidentally the relative frequency and relations of posterior urethritis, prostatitis, epididymitis, and spermatocystitis. The authors have carefully studied 1000 cases of gonorrhœa from this point of view. Their figures appear to show that if posterior urethritis can be prevented these

[8] The Study of Two Hundred and Sixty-four Cases of Gonorrhœal Epididymitis, New York Medical Journal, 1907.

[9] The Prevention of Epididymitis and the Method of Treatment of Gonorrhœa in the Acute and Subacute Stages, Dermatol. Zeitschrift, 1907, xiv, 14.

[10] Pathology and Statistics of Epididymitis, Arch. f. Dermatol. u. Syph., 1908, 89, 255.

[11] Zeitschrift f. Urologie, 1909, iii, 1.

complications hardly occur; also that early recognition and prompt treatment of spermatocystitis should often prevent epididymitis. Of the 1000 cases, 629 had posterior urethritis, that is, 63 per cent., and of this number the prostate alone was inflamed in 385 (61 per cent.); the seminal vesicles (one or both) in 38 (6 per cent.), and the prostate and vesicles together in 180 (29 per cent.). Added together, this makes 565 cases of prostatitis (about 90 per cent.) and 218 cases of spermatocystitis (nearly 35 per cent.). Of the 218 cases of spermatocystitis, 139 were bilateral, 79 unilateral, 47 on the left and 32 on the right side. In the 1000 cases of gonorrhœa were 124 recent cases of epididymitis (12.4 per cent.). With this number were 107 cases of prostatitis, 42 isolated and 65 associated with spermatocystitis. There were 76 cases of spermatocystitis, 65 associated with prostatitis, and 11 isolated. While the authors are not entirely clear on the matter, they give the impression that spermatocystitis is responsible for many cases of subsequent epididymitis. Under the head of treatment, as already said, they agree that early recognition and treatment of it will prevent epididymitis. Of the 218 cases of spermatocystitis, 156 were of the simple superficial or catarrhal type, 50 were examples of chronic inflammation with obliteration fibrosis, and 9 were instances of empyema. Three cases were not accounted for. Of the 371 cases of anterior urethritis alone, there were but 4 with complications, all cases of prostatitis. In this article there are two significant statements: (1) That if posterior urethritis can be prevented, complications hardly occur; and (2) that early recognition and treatment of spermatocystitis will prevent epididymitis. These are in accord with and strengthen my first proposition.

4. Coming now to the question of treatment, it is interesting to note that the effort of most teachers is to simplify it, employing fewer remedies and a more expert procedure. The methods of thirty years ago show an uncertainty and complexity that does not exist today. Not only is therapeutics more effective, but pathology has been very much simplified. Since Neisser's discovery of the gonococcus we have had a definite means of diagnosis, and also a definite means of prognosis of the acute stage. Yet, as already said, no matter what be the form of treatment, the average duration of the acute stage of the disease remains from four to six weeks. Notwithstanding our better understanding of the pathology of the urethra and of the cause of the disease, nature still takes her own time to remedy the results of infection, and to restore to a normal condition, or as near normal as possible, the mucous membrane, which has been devastated. Whether the treatment has been expectant, or by irrigation, or by hand-injection, or by the combination of the expectant and any other method, it seems to take just about so long for a new mucous membrane to be formed. The inference is that our methods should be unirritating, and adapted to

the indications as the latter arise. In 1876 Mr. J. L. Milton, of London, compiled a list of 63 medicaments used for urethral injection, for some of which extraordinary virtues were claimed; such, for instance, as the cure of recent infections in from one to four days, with only two failures in 64 cases! But on the modern definiteness and simpler therapeutics Watson and Cunningham may be quoted. They say: "We do not propose to let ourselves stray from the narrow limits defined by the efficacious remedies which have earned a right to be seriously accepted as having an established value. Those which are worthy to be thus classed are the following: the silver preparations, protargol, argyrol, and nitrate of silver; permanganate of potash, and the astringent remedies, zinc and lead. The first three and permanganate of potash aim at the destruction of the gonococcus, or at inhibiting its activity to the degree that the urethral membrane shall have the power to repair sufficiently to repel its further attacks. The nitrate is of special value in the more chronic stage of the disease."

Notwithstanding the modern attempt at simplification, there is diversity of opinion as to method. In illustration I may make the following citations: Keyes and Keyes, Jr.,[12] say positively that in the previous edition Keyes, Sr., has not advocated irrigation, but that Dr. Chetwood's modification of Janet's method has given results never before obtained in thirty-five years of practice. The results are so much better that he recognizes their obvious superiority, giving Dr. Chetwood the whole credit.

R. W. Taylor[13] recommends zinc injections almost at the onset. Only in the declining stage does he recommend irrigation. In the very acute early stage, when the question of local treatment is a delicate one, he mentions weak permanganate and protargol as antiseptics, stating that they benefit but do not cure. He seems to imply that they may prevent posterior urethritis, but astringents and capsules are his main remedy. Under the paragraph entitled "Fads in the Treatment of Gonorrhœa" he scores the heroic use of antiseptics and ridicules the claims of rapid cure. There may be an apparent rapid improvement, but the discharge is not arrested and the mucosa become succulent, so much so that urination is hindered and bladder irritation develops. Patients seldom try this treatment a second time. He appears to discredit the apparent cures by Janet's method.

Marshall[14] advocates protargol followed by astringent injections as preferable to irrigations, which are irksome. He gives no local treatment if the parts are œdematous. The treatment of chronic urethritis is overdone, chiefly because of the introduction of the

[12] Surgical Diseases of the Genito-urinary Organs, 1903.
[13] Genito-urinary and Veneral Diseases.
[14] Syphilis and Gonorrhœa, 1904.

urethroscope. Patients expect this to be used in all cases, and it is often meddlesome. Instrumentation should be avoided except in very chronic cases.

Hyde and Montgomery (1900), unlike most writers, have a paragraph on prognosis. They regard local treatment as particularly suitable for the stage of decline. Pus is a contraindication. A theory that gonococci are to be killed is responsible for much permanent damage. Weak astringents, if any, should be used. It is best to reserve injections for a patient who has had complications, epididymitis, cystitis, etc.

Fuller[15] speaks well of the newer silver preparations; if used very early in the disease they may prove of much value. Argonin, as introduced by Jadassohn in 1895, he has used considerably. Protargol came out later. It gave good results in private practice where cases are seen early. Janet's method is given exhaustively, but the author believes it causes spermatocystitis, and hesitates to recommend it. He evidently prefers injections of the mild antiseptics when the case can be controlled. Astringents are never to be used until the declining stage, and then not over twice daily.

Morton[16] forbids the use of the astringents before the declining stage. Janet's method will check the purulent discharge in eight days in most cases, but spontaneous relapses occur even in the midst of treatment, and they are often repeated several times. A thin discharge for weeks may persist, so that ultimate recovery is not hastened. This method appears to prevent posterior prostatitis. It must be begun early to be effective. The expense, trouble, and inconvenience are against it, and the author does not appear to advocate Janet's method as a routine procedure; he probably prefers for this purpose the use of silver solutions as antiseptics. Treatment with protargol, 0.25 to 1 per cent., causes improvement in a few days; the acute symptoms subside directly. The protargol is now given in greater concentration, and after a few more days the discharge ceases; but if the protargol is stopped a relapse occurs. The percentage recovering in two or three weeks is small; in most cases five or six weeks are required.

Lydston,[17] in assailing specific and rapid cures for gonorrhœa, says that Janet's method should not be placed arbitrarily in this class. In practice it is very rare for a case to be treated rationally, because both patient and physician underrate the possibilities of the disease. Personal experience of cases is too often based upon simple, mild cases. Local injection is the most available treatment; irrigation requires time and money at best. Proper injections prevent strictures and complications. There is still a popular prejudice against injections; patients are wont to blame them for complications, and

[15] Diseases of the Genito-urinary System, 1900.
[16] Genito-urinary Diseases, 1902.
[17] Surgical Diseases of the Genito-urinary Tract, 1904.

some practitioners, by censuring the injection treatment, conspire with the prejudice. Lydston believes in the modern organic solutions of silver for their bactericidal action, preferring them to astringents, which may impair the defensive activity of the tissues. However, he combines ordinary antiseptics and astringents.

Baumann has a good chapter on prognosis. This in pure gonorrhœal affections is favorable. In subacute and chronic forms it is conditional on several factors—duration, complications, treatment. No method of treatment is free from relapses. Complications are more frequent with chronic gonorrhœa. The author believes nitrate of silver to be the best local remedy. Germicides and antiseptics always irritate; the more antiseptic the more irritant. For irrigation he uses permanganate of potash, 1 to 2000 to 1 to 20,000, and this treatment is not contra-indicated even in the early stages—in fact, he finds it the most beneficial at this time. He also irrigates with nitrate of silver and zinc sulphate in weak concentration.

Greene and Brooks regard posterior urethritis as universally present. They use no local measures until the mucopurulent stage is reached. They object to astringents on theoretical grounds. Mild antiseptics will not injure outright, but are not recommended. Directions however, are given for those who wish to employ them. Then begins the treatment recommended by the authors; it is made to the posterior as well as to the anterior urethra, and irrigation with silver nitrate follows.

Von Zeissl,[18] like Finger, quotes Ricord's aphorism: "We know when the gonorrhœa begins; we know nothing as to when it will end." Many factors affect the prognosis in individual cases. Astley Cooper's dictum is fully borne out today: "In many cases, despite all remedies, the malady lasts so long that it is a reproach to our art." Under "treatment" he speaks of the difficulty of controlling private patients, pointing out that they are exposed to many prejudicial circumstances to which a hospital patient is not. Zeissl laments that the discovery of the gonococcus has not helped us in the treatment. Modern antiseptics, he says, give him no better results than do the older remedies. However, he washes out the anterior urethra with a soft catheter and a permanganate solution. He does this from the start, unless œdema and lymphangitis are present. Protargol may be substituted for the permanganate. He also recommends Janet's method as a later resource. He also uses ordinary injections at short intervals.

Finger seems to be decidedly pessimistic, and apparently does not believe that our methods show any superiority over former ones. On the other hand, he does not assert the contrary. He goes very thoroughly into the history of the treatment, and finds that many of

[18] Frisch and Zuckerkandl's Handb. der Urologie, 1906, 111.

our modern resources are not really new. He finds syringes two hundred years ago differing in no wise from those of today. He also describes the great activity of the specialists of a generation ago in regard to the problem of treatment. He believes in weak protargol injections from the onset, unless œdematous swelling, bloody pus, and phymosis or paraphymosis are present. But as to the uncertain cure and its complications he seems to believe that the disease is the same old unknown quantity it was at the dawn of scientific medicine.

Wassidlow writes in a less pessimistic vein than Finger, but nowhere does he state or imply that our knowledge has progressed in recent years. Neither is the contrary statement made or implied. In a new edition of his book, Casper seems to be as pessimistic as Finger. Under "prognosis" he states that it is doubtful if the majority of cases do not become chronic; while in the chronic stage excesses of any kind may set up acute exacerbations, with all the attending dangers of complications.

Quite a number of writers, however, record their belief that we get better results than formerly. Janet, in his latest article (1907), appears to take a somewhat similar view. On the other hand, all of these continental experts who have been authorities for many years, dating back to pre-irrigation days, do not commit themselves.

In a very interesting article, Streiff[19] shows that irrigation is by no means a new resource. Morgan, of Dublin, employed it in 1869; Durham (Guy's Hospital) in 1870; Windsor, of Manchester, England, in 1871, using permanganate 1 to 1000; Reginald Harrison in 1880; Holbrook Curtis in 1883; Halstead and Van der Poel in 1886; Brewer in 1887, and Reverdin in 1892; all of them preceding Janet; and even earlier than they was Serra, who used irrigation of plain water in 1831. All of these pioneers irrigated the penile urethra alone. Another series of men irrigated the bladder and incidentally the posterior urethra: Cloquer (date not given), Diday in 1839, Reliquei in 1871, Bertholle in 1877, and others. Janet, however, originated modern urethrovesical irrigation, and also the theory that it cured by producing serous reaction and preventing the deep proliferation of gonococci. The author regards Janet's method as a logical development of bacteriology and antisepsis.

Janet's technique has been modified in various ways, and a great number of substances have been substituted for permanganate. In France nearly all surgeons and specialists use the irrigation in some form. They wait for the subsidence of the inflammatory phenomena. Irrigation has some enemies who do not believe in exposing the bladder to possible infection, and who rely upon injections and balsamics.

Streiff says nothing whatever as to the superiority of modern

[19] Old and New Treatment of Urethritis, Thèse de Paris, 1908.

measures. A century ago, or thereabouts, balsamics had superseded local treatment. The latter returned into vogue, but not until the discovery of the gonococcus and antisepsis did it receive its modern endorsement. Streiff claims that the modern treatment of urethritis, including lavage, instillation, use of the endoscope, ointments, sounds, massage, etc., is entirely surgical in its tendency.

My own recollection of what may be termed the era of irrigations in its different stages is still vivid. There was also an era of nozzles for insertion into the meatus urinarius. Many were the zealous experimenters. Everybody devised or modified a nozzle; and every nozzle was provided with both an inlet and an outlet tube, in order to regulate within the urethra the exact pressure of the irrigating fluid. We also "felt our way" with the strength of the germicide, in order to obtain an unirritating and yet efficient solution. At that epoch the bichloride of mercury was an efficient germicide when applied in proper solution to external wounds; would it not be equally efficient in gonorrhœal infection? Acting on this theory, I made an experiment at the City Hospital. I irrigated a considerable number of cases with bichloride of mercury, the solutions beginning with a strength of 1 to 6000. Any one who knows the irritating effect of that solution can imagine the warmth of the reception I received from the patients when I made my next visit to the hospital. The same experimentation was taking place at the Vanderbilt clinic and at the Outdoor Department of Roosevelt Hospital. The result of our combined experience and of our patients' tribulations was to reduce the solution to its proper strength, 1 to 20,000 or 1 to 30,000. Other substances, such as boric acid, hydrastis, methylene blue, and permanganate of potassium were also experimented with.

The house surgeons to a man were enthusiastic over the irrigation treatment, and were eager to employ it in each and every case. When they asked me what antiseptics they should use, I said, "Whichever you please." And now happened a curious thing, to wit, that their choice seemed to be determined in every case by the complexions of the house surgeons. I will not say *post, ergo propter*, but, as a matter of fact, all the blond men chose to experiment with plain white solutions or with methylene blue, while the brunette men invariably treated their suffering patients with the red permanganate of potassium. I leave the explanation of this to the metaphysicians.

My own conclusions in regard to the irrigation method were that it did not readily control the symptoms, but, on the contrary, that it was even liable to aggravate them. In many cases of acute gonorrhœa the patients can hardly tolerate their own urine passing through the urethera; therefore the introduction from without of a fluid which, even with the most watchful care, may overdistend the canal, is liable to cause traumatisms and aggravate the conditions. Fur-

thermore, as time went on I became convinced that these irrigations not only did not shorten nor mitigate the attack, but that posterior urethritis was more prevalent, and that therefore inflammation of the contiguous structures, the prostate, the epididymis, etc., was likelier to occur. Moreover, the irrigations were inconvenient, they were sloppy, and because of the time required were difficult to carry out. Consequently, for acute gonorrhœa a change was made from that method to the one I now prefer. In chronic states irrigations may have their place; but even then only in exceptional cases.

There is a method called by the Germans the "provocative method." Zieler,[20] chief of Neisser's clinic at Breslau, who evidently represents the opinion of many of his colleagues, believes that whatever cures gonorrhœa can do so only by exciting hyperemia and serous transudation. The "inflammatory serum," as he terms it, is fatal to the deep proliferation of gonococci, which tend to return to the surface, where also many have been present from the beginning. In these more exposed situations they may be destroyed by antiseptics. The benefits of irrigation he attributes solely to the hyperemia set up mechanically, not at all to the permanganates. Protargol, argyrol, etc., are both hyperemizing and antiseptic, hence the good results from their use. The old-fashioned astringents are contra-indicated because they antagonize the hyperemic tendency and permit the deep proliferation of gonococci.

German physicians use the expression "provocative treatment" for the use of mechanical or chemical irritants intended to cause hyperemia, transudation, and destruction of gonococci. A writer having termed Dr. Carl Alexander's (Breslau) hydrogen peroxide treatment a "provocative" measure, the latter replies,[21] stating that his (1 per cent.) injection or irrigation does not belong under this head. He uses it to oxidize and destroy the gonotoxin. Further, the liberation of gas in the urethra exerts a mechanical action on foreign material, but not an irritating one. He regards his procedure as an addition to our resources; if it fails now and then, so do all methods. My own experience with hydrogen peroxide was not at all satisfactory; though it was not used by irrigation, which I agree with Kreissl[22] is a step backward, and gives poor results, tending to complications and chronicity. With the advent of the albuminoid salts of silver, we have had at our disposal better means of controlling the infection. Especially if used in the early stages of the disease, they will, in a large proportion of the cases, modify its progress and lessen the liability to complications. Their use by the hand-injection method, followed by such other means as may be indicated in the later stages of the malady, will meet all the requirements. The salts of silver are well under control; they

[20] Münch. med. Woch., 1907, 305.
[21] Zeitschrift f. Urologie, 1909, iii, 1.
[22] Urogenital Therapeutics, Chicago, 1908.

can be used in the most intolerant urethra without aggravating the patient's condition, and they can be placed in the hands of the patient himself. But it must not be overlooked that the latter must also be *treated.* Doubtless you will think it a mere truism to say that a patient's habits, social condition, etc., affect his vital processes. But we must take advantage of whatever physiology can do to limit the supply of pabulum to the infection and assist in strengthening the resistance of the tissue, and therefore it is as important to consider the patient's environment as to give him local treatment.

To me a very important fact is that the human urethra is now looked upon with what might be termed greater respect. This is not merely a speculative statement. It has a practical application. The old-time, coarser point of view, that the urethra was a mere "water pipe," and that if an individual subjected himself to conditions which infected this conduit he was the victim of his own folly, and, to use the common phrase, "it served him right." This view naturally tended to coarse and unsympathetic treatment. But it is now recognized that the uerthra is a delicate, highly endowed organ, susceptible to grave local damage, and that its infection may be propagated to distant, even to vital organs, and to innocent persons. Based upon this, together with a knowledge of the specific cause of the disease our methods have become more definite, and our technique more delicate and gentle. Therefore there is ground for the statement that the average case shows less tendency to become chronic; and with our ability to inhibit the activity of the infection when the case is seen early there is less likely to be a posterior urethritis and, therefore, less liability to infection of the contiguous structures.

The subject is a large one, and much remains to be accomplished, yet, notwithstanding the dubious tone of the literature which I have tried to review I am satisfied that real progress has been made in the treatment of gonorrhœa.

HELMINTHIASIS IN CHILDREN.

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The study which it is the object of this paper to record was undertaken in the effort to determine: (1) The frequency with which children between the ages of two and twelve years harbor intestinal worms; (2) the species of parasites harbored and the relative frequency of their occurrence; (3) the number of cases in which the common

intestinal worms are responsible for symptoms, and the nature of the symptoms produced; and (4) the occurrence and significance of eosinophilia in infections with intestinal worms. These investigations were based on the discovery of intestinal worms, their parts, or their ova in the feces. Whenever possible, the parasite was obtained after treatment, and in positive cases the blood was examined to determine the percentage of hemoglobin and the percentage of the eosinophile cells. The technique used in making the examinations will be given in some detail under the general discussion of diagnosis.

In this paper it is only intended to discuss the results of these investigations and to consider phases of the subject of helminthology which have a practical medical bearing. No attempt has been made to review the enormous literature, but work will only be cited which has a bearing on the investigations or is not to be found in the usual text-books. The investigations were conducted upon 310 children between two and twelve years of age. For purposes of convenience and accuracy the cases have been divided into two groups. The first group comprises 30 cases, the second 280.

The first group of thirty examinations were made entirely on the basis of suspicious symptoms, and were in no way consecutive; hence they are of little statistical value. This portion of the work extended over a period of four months, and the children examined suffered from obscure nervous or gastro-intestinal disorders, which were not explained by the history or physical examination. This group also includes 4 cases in which the parasites had been seen previous to admission.

As shown in Table I, twelve of the children in this group harbored intestinal worms, and, with the exception of one case (VII), the relationship of the symptoms to the presence of the parasite is shown by the influence of treatment. In the case mentioned the child disappeared from observation before treatment could be instituted, and in consideration of the fact that the parasite harbored rarely produces symptoms, this case must be considered doubtful. Three other cases (IV, V, and XII) were lost track of after treatment was begun, but the nature of the symptoms and the improvement with treatment give sufficient indication that the intestinal worms were the causative agency. The parasites found in the 12 positive cases were as follows: Ascaris lumbricoides in 2 cases, Trichuris trichiura in 2 cases, and Oxyuris vermicularis in the remaining 8 cases. The symptomatology and blood examinations will be dealt with under the headings of the different parasites.

The second group comprises 280 examinations, which were made as nearly consecutive as possible, on all children within the prescribed age limits from families whose members were under treatment at the clinics. These investigations extended over a period of thirteen months. From this group of 280 children, 80 (28.57

TABLE I.—POSITIVE CASES FROM 30 EXAMINATIONS MADE ON PATIENTS WITH SUSPICIOUS SYMPTOMS.

No.	Sex and age.	Nationality.	Parasites.	Symptoms.	Blood Examination. Polynuclear cells. Per cent.	Lymphocytes. Per cent.	Large mononuclear and transitional cells. Per cent.	Eosinophiles. Per cent.	Basophiles. Per cent.	Hemoglobin. Per cent.	Result.
1	Male 3½	U. S.	Ascaris lumbricoides	Fever. Temperature, 101° to 102° F. at night for ten days. Very restless during sleep, frequently cried out as if in pain. Frequent vomiting. Loss of appetite. Constipation. Sudden onset of illness	36.0	40.0	10.1	12.1	0.2	70.0	Recovery after expulsion of worm.
2	Male 3½	Parents German. U. S.	Ascaris lumbricoides	Ill three months. Irritable, loss of appetite, poor color, loss of weight. Jaundice one week. Passed roundworm four months ago	40.8	30.3	16.1	12.6	0.8	65.0	Improvement after expulsion of two worms.
3	Male 4	Father German. Mother U. S U. S.	Oxyuris vermicularis	Loss of weight. Attacks of pain in lower portion of abdomen. Genital and nasal pruritis. Restless at night. Appetite poor. Worms seen by mother	40.0	39.1	13.2	7.0	0.6	75.0	Improvement with treatment. Recovery in one month.
4	Female 5	U. S.	Oxyuris vermicularis	Rectal irritation at night. Sleep interrupted. Irritable. Poor appetite. Constipation. Worms seen by mother	58.1	18.2	13.0	8.0	1.0	—	Improvement for one week. Lost track of.
5	Female 6	U. S.	Oxyuris vermicularis	Genital pruritis. Masturbation. Vulvitis. Loss of appetite, slight loss of weight. Mother not aware of infection	53.2	25.0	18.3	2.1	0.5	60.0	Improvement for ten days. Disappeared from observation.
6	Female 7	Russia	Oxyuris vermicularis	Nasal and genital pruritis. Loss of appetite, loss of weight. Mother not aware of infection	60.0	21.2	14.0	3.3	0.1	60.0	Recovery with treatment.
7	Male 7	U. S.	Trichuris trichiura	Loss of weight, anemia, loss of appetite	40.0	30.3	24.0	4.0	1.2	55.0	Lost track of. Treatment not given.
8	Female 7	U. S.	Oxyuris vermicularis	Loss of weight and appetite. Sleeplessness, night cries. Nasal pruritis. Had passed round worm. Mother not aware of threadworm infection	52.2	24.0	10.0	12.6	0.4	—	Improvement with treatment. Complete recovery in five weeks.
9	Male 7	U. S.	Trichuris trichiura	Anemia, loss of weight. Puffiness of lower eyelids. Appetite, sleep, and bowels normal	Red blood cells, 3,100,000 per c.mm. White blood cells, 6000 per c.mm. Nucleated red cells (normoblasts) 43.6	40.0	16.0	0.3	—	40.0	Gradual improvement with treatment. Hemoglobin, 65 per cent. at end of eight weeks.
10	Female 8	U. S.	Oxyuris vermicularis	Restless at night. Rectal irritation. Appetite normal. Worms seen by mother	40.3	35.0	18.3	6.0	0.1	75.0	Recovery.
11	Female 8	U. S.	Oxyuris vermicularis	Headache, sleeplessness, night cries. Slight loss of weight, poor appetite, constipation. Irritable. Mother not aware of infection	Red blood cells, 4,100,000 per c.mm. White blood cells, 16,000 per c.mm. 29.2	26.1	11.0	33.0	0.6	70.0	Complete relief of symptoms after one week.
12	Female 8	U. S.	Oxyuris vermicularis	Irritable. Rectal irritation, genital pruritis. Appetite capricious, bowels constipated. Worms seen by mother	56.0	24.1	13.0	6.2	0.3	75.0	Improvement. Relief of symptoms in one week. Lost track of.

per cent.) harbored intestinal worms. Five of these 80 children were infected with two species of parasite, which gives a total of 85 infections. Thirty-one (11.07 per cent.) of the children were infected with Trichuris trichiura, 23 (8.21 per cent.) harbored Oxyuris vermicularis, 20 (7.14 per cent.) were infected with Hymenolepis nana, 6 cases (2.14 per cent.) harbored Ascaris lumbricoides, and Tenia saginata was found in 5 cases (1.78 per cent.).

In the double infections, Hymenolepis nana and Trichuris trichiura were present together in 2 cases, Hymenolepis nana and Oxyuris vermicularis in 1 case, and Ascaris lumbricoides and Oxyuris vermicularis were associated in 2 cases. Out of the total of 85 infections, Trichuris trichiura occurred in 36.47 per cent., Oxyuris vermicularis in 27.05 per cent., Hymenolepis nana in 23.52 per cent., Ascaris lumbricoides in 7.05 per cent., and Tenia saginata in 5.88 per cent.

The only recent statistical study of the intestinal worms of children in this country that I have been able to find is that of Stiles and Garrison.[1] These investigators examined the feces of 123 children under fifteen years of age, and found evidence of infection with intestinal worms in 26 cases (21.14 per cent.). Trichuris trichiura was present in 16 cases (13.01 per cent.), Oxyuris vermicularis in 2 cases (1.63 per cent.), Ascaris lumbricoides in 1 case (0.81 per cent.), and Hymenolepis nana in 6 cases (4.88 per cent.). There were no cases of infection with Tenia saginata in children.

The important features of my cases will be considered under the headings of the different parasites.

Trichuris trichiura (Trichocephalus dispar, T. hominis, T. trichiura, the whipworm). Table II; Cases VII and IX, Table I. In the first group of examinations (Table I) there were 2 instances of infection with this parasite; in the second group, 31. In 2 cases of the second group this parasite was found in association with Hymenolepis nana.

Symptomatology. None of the cases in the second group presented symptoms due to the whipworm, and 1 of the 2 cases in the first group must be excluded, since the relationship of the symptoms to the presence of the parasite is unproved. There seems little doubt that the symptoms in the other case (IX, Table I) were due to the presence of the parasite. This patient, a boy, aged seven years, had lost weight for nearly a year, and during this time he had become pale and listless. Appetite, sleep, and bowel movements were normal. There was slight puffiness of the lower eyelids, and the blood showed a moderate grade of secondary anemia with the presence of nucleated red cells (normoblasts). The number of red cells was 3,100,000 per cubic millimeter, and the hemoglobin 40 per cent. The urine was negative. The usual tonics (iron, arsenic,

[1] Bull. No. 28, Hyg. Lab. U. S. Pub. Health and Marine Hosp. Serv., 1906.

and cod-liver oil) had been administered over a period of four months, with little benefit. Numerous ova of the whipworm were discovered in the feces, and the patient was treated on the basis of this finding. Twice a week for three weeks thymol (12 grains) was given in divided doses, and during this time the feces were frequently examined to determine the number of ova. After the first two treatments the number of ova greatly diminished, and then remained stationary. Irrigations of salt water, quassia, and garlic infusions were given with little appreciable effect. Benzine irrigations were given according to the recommendation of Hemmeter,[2] with the result that the feces became free from ova. No other treatment was used, and no change was made in the mode of life. The improvement was marked. At the end of eight weeks the patient had gained three and a half pounds and the hemoglobin had risen to 65 per cent. Owing to the distance at which the patient lived it was impossible to examine the feces to determine the number of parasites expelled. The mother, however, noted the parasites, and specimens were brought for verification.

This case is given in some detail, because the whipworm is in most instances a harmless parasite. In some cases, however, when present in large numbers, this parasite may give rise to severe symptoms, and may even cause death. This is not surprising, since Askanazy[3] has shown that the intestinal canal of this parasite contains blood pigment, and Guiart and Garin[4] found that the stools of those infected reacted positively to the Weber test for occult blood. Becker[5] has collected cases from the literature in which the whipworm was responsible for definite symptoms. The symptoms were frequently intestinal: diarrhœa, often with bloody stools, vomiting, abdominal pain, etc. In other cases nervous symptoms, such as dizziness and severe headaches, occurred. Becker reported a case of secondary anemia which closely resembles the case cited. Theodor[6] reported a case of progressive pernicious anemia in a boy, aged eleven years, whose stools contained numerous ova of T. trichiura. Somewhat similar cases have been reported by Ostrovsky[7] and by Sandler.[8]

Blood Examinations.[9] The published reports on this subject

[2] Diseases of the Intestine, 1902, p. 582.
[3] Deut. Archiv f. klin. Med., 1896, lvii, 104.
[4] Semaine méd., xxix, No. 35.
[5] Deut. med. Woch., June 26, 1902, p. 648.
[6] Archiv f. Kinderheilk., 1900, xxviii.
[7] Abst. New York Med. Jour., 1900, lxxii, 826.
[8] Deut. med. Woch., 1905, xxxi, 95.
[9] The differential counts were made from blood smears stained with Wright's stain, and 500 to 1000 cells were counted. Most of the hemoglobin estimations were made with the Sahli hemometer. The instrument used had been standardized by the blood of normal children. The average percentage of hemoglobin in children between two and six years was 70 to 80 per cent; in children six to twelve years of age, 75 to 85 per cent. A few of the hemoglobin estimations were made with the Talquist scale, which I have found to give readings approximately the same as the Sahli instrument. When counts of the blood cells were made, the Thoma-Zeiss ap aratus was used.

TABLE II.—TRICHURIS TRICHIURA CASES FROM 280 CONSECUTIVE EXAMINATIONS.

No.	Sex and age.	Nationality.	Parasites.	Symptoms.	Blood Examination.						Result.	
					Polynuclear neutrophile cells. Per cent.	Lymphocytes. Per cent.	Large mononuclear and transitional cells. Per cent.	Eosinophile cells. Per cent.	Basophile cells. Per cent.	Hemoglobin. Per cent.		
1	Female 3	U. S.	....	None							....	....
2	Male 3	Russia	....	None	No apparent leukocytosis						....	....
3	Male 4	U. S.	....	None	52.3	31.5	12.0	3.5	0.5	60.0	....	....
4	Male 4	U. S.	....	None	44.1	37.3	15.0	2.1	0.5	70.0	....	....
5	Male 5	U. S.	....	None	No apparent leukocytosis						....	....
6	Female 5	U. S.	....	None	51.0	28.0	16.0	3.1	0.6	70.0	....	....
7	Male 5	Italy	....	None	Slight leukocytosis (?) (estimated)						....	....
8	Male 6	U. S.	....	None	49.0	32.7	16.5	1.7	—	60.0	....	....
9	Female 7	U. S.	....	None	No apparent leukocytosis						....	....
10	Female 7	Ireland	....	None	55.5	20.6	19.1	4.5	0.1	80.0	....	....
11	Female 7	U. S.	....	None	No apparent leukocytos.s						....	....
12	Male 7	U. S.	....	Double infection, H. nana and T. dispar. No symptoms referable to helminthiasis. See No. 15, Chart IV	46.0	41.2	9.2	3.5	—	75.0	....	....
13	Female 8	U. S.	....	None	No apparent leukocytosis						....	....
14	Male 8	U. S.	....	None	51.0	34.1	10.0	2.8	—	75.0	....	....
					No apparent leukocytosis						....	....
					49.1	22.4	24.0	3.2	0.5	80.0	....	....

15	Male 8	U. S.	....	None								....	....
16	Male 8	Ireland	....	None	Slight leukocytosis (?) (estimated)							....	....
					53.4	24.1	16.8	4.4	0.8	70.0			
17	Male 8	U. S.	....	None								....	....
18	Female 9	U. S.	....	None								....	....
19	Female 10	Germany	....	None	65.3	19.2	10.0	4.9	—	75.0		....	....
20	Male 10	U. S.	....	None	No apparent leukocytosis							....	....
					63.0	20.0	13.5	3.2	—	70.0			
21	Male 10	U. S.	....	None	No apparent leukocytosis							....	....
					55.5	19.8	22.0	2.2	0.4	65.0			
22	Female 10	Sicily	....	Double infection, H. nana and T. dispar. No definite symptoms. See No. 19, Chart IV								....	....
23	Male 10	U. S.	....	None	No apparent leukocytosis							....	....
					44.3	35.0	18.0	1.9	0.3	65.0			
24	Male 10	U. S.	....	None								....	....
25	Male 10	U. S.	....	None								....	....
26	Female 11	U. S.	....	None								....	....
27	Male 11	U. S.	....	None	No apparent leukocytosis							....	....
					46.1	33.2	13.2	6.4	0.9	65.0			
28	Male 12	U. S.	....	None	No apparent leukocytosis							....	....
					65.0	20.0	12.0	2.4	0.4	75.0			
29	Female 12	U. S.	....	None								....	....
30	Female 12	U. S.	....	None	70.0	19.2	6.0	4.1	0.1	75.0		....	....
31	Male 12	U. S.	....	None								....	....

indicate that the whipworm rarely produces an increase in the percentage of the eosinophile cells. In the case reported by Becker the eosinophile cells were 2 per cent. French and Boycott[10] made differential blood counts on 26 patients who harbored this parasite, and found that the eosinophile cells were not increased. One case showed 5.5 per cent. of eosinophile cells, but even though 5 per cent. is taken as an arbitrary standard, this single observation is of little importance. Brown[11] mentions that in no less than 10 or 12 cases in which Trichocephalus hominis appeared alone, the percentage of eosinophile cells rarely fell below 5. No cases are cited, nor are the exact percentages of the eosinophile cells given. Naegeli[12] has found eosinophilia in whipworm infections. Manson[13] says that in some few cases eosinophilia was found in persons infected with this parasite, but that this does not appear to be the rule. I have made differential counts in 18 cases of single infection with the whipworm, and in only 1 case was the percentage of eosinophile cells above 5 (XXVII, Table II). In this case the eosinophile cells were 6.4 per cent. Two other children from the same family harbored the threadworm, and it is quite possible that the patient in question was infected with threadworms, which were not detected in the routine examination.

Treatment. It is fortunate that this parasite so rarely causes symptoms, for the treatment is notoriously unsatisfactory. Stiles[14] cites an instance in which 300 parasites were expelled by thymol. In experimental infections in dogs Stiles and Pfender found thymol of little value. In the single case in which treatment was given by me, the number of parasites seemed to decrease under treatment with thymol, judging by the number of ova found in the stools, and the ova finally disappeared with the use of benzine irrigations.

Oxyuris vermicularis (the threadworm, pinworm, or seatworm). Table III; Cases III, IV, V, VI, VIII, X, XI, and XII, Table I. In the consecutive examinations there were 23 cases of infection with this parasite, but I am of the opinion that more children harbor this worm than is indicated by these figures. It is well known that the ova of this parasite are not frequently found in the feces, but that the female worms are more often present. It is possible that when only a comparatively small number of worms are harbored they may be passed intermittently, and in consequence not be found in a single examination. Moreover, the small specimens of feces obtainable for these examinations may not have contained the worms, even though they were being passed at the time. Although in most instances a calomel purge was given before

[10] Jour. Hyg., 1905, v, 274.
[11] Bost. Med. and Surg. Jour., cxlviii, 583.
[12] Blutkrankheiten u. Blutdiagnostick, von Veit, Leipzig, 1908.
[13] System of Medicine, Albutt andRolleston, 1907, vol. ii, part ii, p. 908.
[14] Modern Medicine, Osler and McCrae, 1907, i, 604.

obtaining the specimens of feces, three of the charted cases show how easily these worms may be overlooked. In Case IV, Table III, the presence of this worm was not suspected, but two pregnant female oxyurides were found in the feces after treatment for H. nana. Similarly, in Cases V and XVI, threadworms were discovered in the feces after the administration of santonin in the treatment for ascaris. The autopsy records of Still[15] are interesting, as showing the frequency of this worm. Out of 200 consecutive autopsies performed at the Great Ormond Street Hospital, in London, Still found the threadworm in 32 of 100 children between two and ten years of age.

Symptomatology. In 6 cases from the second group of examinations the threadworm was present without giving rise to symptoms (I, IV, V, VII, XV, and XVI, Table III). In one of these cases (XV) the child had previously suffered from symptoms, but none were present at the time of examination. The irritative symptoms produced by the nocturnal wanderings of these worms usually leads to their detection, but when local symptoms (rectal irritation, genital pruritus, etc.) are absent, the infection may not be suspected. In 4 cases from the first group of examinations, and in 5 of 17 children from the second group, who suffered from symptoms, the mother was not aware of the infection.

The most frequent symptoms referable to the threadworm are those of irritative nature due to the migration of the pregnant female worms. In this class is the genital pruritis and the rectal irritation. The vulvitis and masturbation in Case V, Table I, were probably of this origin, and both of these symptoms disappeared after appropriate treatment. Loss of weight, anemia, and headache are not infrequent symptoms, and may form the complaint for which the child is brought to the physician. This fact has been pointed out by Still.[16] The reflex nervous disturbances produced by this parasite are of importance. Restlessness at night, grinding of the teeth, night cries, and general irritability are particularly frequent. Cases are reported in which the threadworm may be responsible for convulsions or choreiform movements. Holt[17] cites a case in which threadworms were the probable cause of chorea. Gastro-intestinal symptoms are rather common. Still[18] has called particular attention to pain in the lower part of the abdomen and right iliac fossa as a symptom in Oxyuris infections. Sometimes the pains are referred to the umbilical region. Ashhurst[19] has recently reported a case in which, on operation for appendicitis, the appendix was found to contain numerous oxyurides. Culhane[20] has reported

[15] Brit. Med. Jour., 1899, i, 898.
[16] Common Disorders of Childhood, 1909.
[17] Diseases of Infancy and Childhood, 1909.
[18] Common Disorders of Childhood, 1909.
[19] Amer. Jour. Med. Sci., October, 1909, p. 583.
[20] Jour. Amer. Med. Assoc., 1910, liv, 48.

TABLE III.—CASES OF INFECTION WITH OXYURIS VERMICULARIS. FROM 280 CONSECUTIVE EXAMINATIONS.

No.	Sex and age.	Nationality.	Parasites.	Symptoms.	Blood Examination. Polynuclear neutrophile cells. Per cent.	Lymphocytes. Per cent.	Large mononuclear and transitional cells. Per cent.	Eosinophile cells. (Per cent.	Basophile cells. Per cent.	Hemoglobin. Per cent.	Result.
1	Male 3	U. S.	....	None due to helminthiasis. Mother not aware of infection	No apparent leukocytosis 46.7	32.2	16.2	4.2	0.3	79.0	
2	Female 3	U. S.	....	Restless at night, grinds teeth. Intense itching of genitals and nose. Very pale, poor appetite, bowels normal, puffiness of lower eyelids. Mother has noticed worms for three weeks	Moderate leukocytosis 27.7	47.4	14.6 (estimated)	8.7	0.3 myelocytes 0.1	60.0	Recovery.
3	Female 3	U. S.	....	Restless at night, genital and nasal pruritis. Appetite and bowel movements normal. Child pale and thin. Mother has seen worms at intervals for one year	No apparent leukocytosis 36.0	46.3	16.6	0.95	—	65.0	Recovery.
4	Male 4	U. S.	....	Double infection, Oxyuris vermicularis and H. nana. Symptoms probably due to latter. See No. 9, Chart IV							Two threadworms were found in the feces after treatment for H. nana.
5	Male 4	U. S.	....	Double infectiona, scaris and Oxyuris vermicularis. Symptoms probably due to former. See No. 1, Chart V							One threadworm found in feces after treatment for A. lumbricoides.
6	Male 4	U. S.	....	Symptoms at intervals for three years. Genital and nasal pruritis. Night cries. Child pale and thin. Appetite poor when symptoms are present. Bowels regular. Mother aware of infection	No apparent leukocytosis 37.3	44.1	15.0	2.1	0.5	55.0	Improvement. Lost track of after twelve days.
7	Male 5	Italy	....	No apparent symptoms	No apparent leukocytosis 44.6	38.1	13.0	3.2	0.2	—	
8	Female 5	U. S.	....	Child pale, slight loss of weight, night cries. Appetite capricious, bowels regular. Mother not aware of infection	Blood examination not obtained						Lost track of. Treatment not given.
9	Female 5	U. S.	....	Has not been well in two years. Flatuency; belches much gas after meals. Nausea. Cries out and jumps during sleep. Appetite poor, bowels normal. Mother has seen worms at intervals during two years	Red blood cells, 3,600,000 per c.mm. White blood cells, 5000 per c.mm. Thoma-Zeiss apparatus 57.5	27.7	14.0	0.2	0.5	40.0	Improvement with treatment. After two weeks symptoms had almost disappeared, hemoglobin, 50 per cent. Hemoglobin, 60 per cent. one month later.
10	Female 5	Ireland	....	Rectal irritation, genital and nasal pruritis. Restless at night, grinds teeth. Appetite and bowels normal. Mother has seen worms for three weeks	Slight leukocytosis (estimated) 36.1	42.0	13.4	7.1	0.2	65.0	Recovery.

11	Female 5	U. S.	....	None referable to helminthiasis. Mother had not seen worms	No apparent leukocytosis	69.3	14.8	13.1	1.9	0.6	70.0		
12	Male 6	U. S.	....	One month, itching of nose and genital region. Very restless at night, grinds teeth. Appetite and bowels normal. Mother had seen worms	No apparent leukocytosis	50.4	26.2	12.2	9.0	0.3	—	Improvement with treatment.	
13	Male 6	U. S.	....	Restless at night. Rectal irritation, genital and nasal pruritis. Appetite excessive. Bowels normal. Mother had seen worms	Slight leukocytosis (estimated)	26.4	49.0	13.2	10.5	0.6	—	Recovery.	
14	Male 6	Ireland	....	Restless at night, night cries. Occasional attacks of right-sided abdominal pain. Bowels normal. Appetite capricious. Mother has seen worms for three weeks	No apparent leukocytosis	39.3	40.5	11.0	6.1	1.2	70.0	Relief of symptoms by treatment.	
15	Female 6	U. S.	....	No symptoms at time of examination. Mother noticed worms six months previously, at which time treatment was given for three weeks	Blood not examined								
16	Female 6	U. S.	....	Double infection, A. lumbricoides and O. vermicularis. Child pale and sparely nourished. No definite symptoms	No apparent leukocytosis	42.0	40.2	14.3	1.8	0.5	70.0	Two oxyurides discovered in the feces after treatment for ascaris.	
17	Male 7	Russia	....	Loss of weight. Pale. Child very fidgety, irritable. Attacks of nausea, no vomiting. Appetite capricious, bowels regular. Mother not aware of infection	Blood not examined							Relief of symptoms by treatment. Time of observations, three weeks.	
18	Female 7	U. S.	....	For one month has complained of slight pain in right iliac fossa. Loss of appetite, loss of weight. Mother not aware of infection	Number of leukocytes apparently normal	60.3	16.1	12.0	9.3	1.0	60.0	Recovery.	
19	Male 7	Germany	....	Restless at night, night cries. Very nervous and irritable. Constipated, appetite poor. Worms observed by mother	Leukocytes 16,000 (estimated)	60.1	18.0	6.0	13.1	2.0	70.0	Recovery.	
20	Male 7	U. S.	....	Restless at night, grinds teeth. Genital pruritis. Appetite normal, bowels regular. Mother not aware of infection	Slight leukocytosis (estimated)	55.9	17.6	17.6	7.4	0.9	—	Lost track of.	
21	Female 8	U. S.	....	Restless at night. Rectal irritation, genital and nasal pruritis (intense). Occasional abdominal pains. Appetite poor, bowels regular. Worms seen by mother	Moderate leukocytosis (estimated)	46.0	16.0	22.3	14.0	1.1	65.0	Recovery.	
22	Male 11	U. S.	....	Nauseated after eating for one month. Frequent headaches. Bowels regular. Appetite poor. Mother not aware of infection	No apparent leukocytosis	50.0	22.0	20.3	7.1	0.03	70.0	Relief of symptoms after two weeks treatment. Lost track of.	
23	Male 11	U. S.	....	Talks in sleep. Occasional night cries. Appetite excessive. Genital pruritis. Worms seen by mother. Has been troubled with threadworms off and on for three years	No apparent increase in number of leukocytes	45.0	39.0	9.7	6.0	0.2	65.0	Improvement for three weeks. Lost track of.	

a similar case, and considers that the oxyurides were the cause of the appendicitis. Still[21] noted thickening and swelling of the appendix in some of his autopsies.

Pains in the lower part of the abdomen or right iliac fossa were present in 4 of my cases. The pains caused considerable annoyance, and in 1 case were of moderate severity. In these cases the temperature was normal and tenderness on palpation or muscle spasm were never elicited. The appetite was poor in 11 cases, capricious in 5, excessive in 2, and normal in 7 cases. In 1 case flatulency and nausea were complained of; in another, nausea alone. Diarrhœa, with blood and mucus in the stools, may be produced by this parasite. Diarrhœa was not a symptom in any of my cases, but constipation was frequent.

Blood Examinations. Differential blood counts were made on 22 patients who suffered from symptoms due to Oxyuris vermicularis, and in 17 the eosinophile cells were above 5 per cent. The percentage of the esoinophile cells varied greatly, and was between 6 and 10 per cent. in 12 cases, between 10 and 20 per cent. in 4 cases, and above 20 per cent. in 1 case. The eosinophile cells were not increased in 5 of the cases showing symptoms. Three of these cases (III, VI, and IX, Table III), from the second group of examinations, gave a history of infection with the threadworm for one, three, and two years, respectively, and the hemoglobin percentage in all 3 was low. Case IX showed a moderate grade of secondary anemia, with a red cell count of 3,600,000, a leukocyte count of 5000, and a hemoglobin percentage of 40. In Case III the hemoglobin was 65 per cent., in Case VI it was 55 per cent. In the two cases from the first group of examinations (V and VI, Table I) it was impossible to determine the duration of infection. In both cases the hemoglobin percentage was low. One patient, however (XXIII, Table III), gave a history of an infection of three years' duration, and the eosinophile cells were 6 per cent. Differential blood counts were made on 3 patients who did not suffer from symptoms, none of which showed an increased percentage of eosinophile cells.

Case VIII, Table I, is of particular interest, since this child was seen at the height of the infection, and the percentage of the eosinophile cells was followed during treatment. This patient suffered from rather pronounced symptoms, and at the time of admission the eosinophile cells were 33 per cent., the red blood cells 4,100,000 per cubic millimeter, the leukocytes 16,000 per cubic millimeter, and the hemoglobin 70 per cent. After treatment for five days the symptoms were much less severe, and the eosinophile cells were 13 per cent. Two weeks later the patient was free from symptoms, and at this time the eosinophile cells had fallen to 3 per cent.

[21] Brit. Med. Jour., 1899, i, 898.

In Boycott's[22] cases the eosinophile cells were above 5 per cent. in 8 out of 18 cases of threadworm infection in children. In the remaining 10 cases the eosinophile cells were not increased. No mention is made of the presence or absence of symptoms, but this investigator suggests that the presence of eosinophilia bears relation to the duration of infection.

Treatment. There are several observations, not generally recognized, which have direct bearing on the treatment for this parasite. An experiment of Grazzi[23] shows that the worms may arrive at maturity in the intestines during the last four or five weeks following a single infection. Since it is probable that fresh parasites constantly develop through auto-infection, the treatment should be continued for six weeks.

In the autopsies of Still[24] the worms were found in the appendix in 25 of the 38 cases which harbored Oxyuris vermicularis; in 6 cases the appendix seemed to be the only habitat.

The normal habitat of this worm is the cecum or appendix, and not the rectum and colon, as often stated. When the females become impregnated they migrate to the lower portions of the large intestine to discharge their ova. They often wander through the rectum and may pass out with the feces. Thus, the treatment should be given with two aims: first, to remove the worms which have migrated to the large intestine; and next, to expel those in the cecum or appendix. For the former purpose the usual irrigations of salt water, quassia, garlic, etc., are effective. As fluid injected per rectum may not always reach the cecum, internal treatment is of importance. Santonin is probably the most useful drug for this purpose, and is best given in doses of 1 to 3 grains, with the same amount of calomel, for three successive evenings. On the first and third mornings of treatment a cathartic should be given. This treatment may be repeated two or three times during the first three weeks.

During the first two or three weeks the irrigations should be given each evening, and from 6 to 20 ounces—depending on the age of the patient—can be given in each injection. Later, the irrigations may be given every alternate evening, and finally twice a week. Every effort should be made to prevent auto-infection, as this is a potent factor in keeping up the disease. At night a mild mercurial ointment (10 to 20 per cent.) may be applied around the rectum. The child should be prevented from scratching and from putting his fingers into his mouth.

A review of the tabulated cases shows how unsatisfactory the usual treatment may be. The mothers usually give the irrigations only during the period of active symptoms, or while worms are passed;

[22] Brit. Med. Jour., 1903, ii, 1267.
[23] Quoted by Manson, System of Medicine, Allbutt and Rolleston, 1907, vol. ii, part ii, p. 891.
[24] Brit. Med. Jour., 1899, i, 898.

often within two to four months the patients again show signs of severe infection. Although this parasite rarely, if ever, produces dangerous symptoms, yet the continual irritation which they set up may undermine the general health; this serves as a sufficient indication for thorough treatment.

Hymenolepis nana (Tenia murina, Tenia nana, the dwarf tapeworm). Table IV.[25] There were 20 cases, of infection with this parasite out of the 280 consecutive examinations. Nineteen of the patients were born in New York City; 1 patient was born in Sicily, and came to this country at the age of four years. This may have been an imported case, as the dwarf tapeworm is a comparatively common parasite in certain parts of Sicily.

Previous to the paper of Stiles,[26] in 1903, the dwarf tapeworm was not considered a common American parasite, but since this time a number of cases have been recognized in different sections of the country. In my investigations this parasite was the third in frequency, and there is every indication that it is a comparatively common, though perhaps unrecognized, parasite of children.

Seventy-nine, or 74.52 per cent., of the cases collected by Ransom[27] in 1904 were in children, and this parasite has been generally recognized as occurring most frequently in individuals under sixteen years of age.

No attempt will be made to give a description of the parasite, as this has been done in another paper. The dwarf tapeworm possesses certain points of similarity to the larger tapeworms, but differs in its minute size and the great number of the parasites usually present. The average length of the parasite ranges below 20 mm. (0.8 inch), and the worm contains from 110 to 200 segments. The number of parasites present in a single patient varies from a few to thousands. After treatment, 50 worms were recovered from the feces of one of my cases, and 60 from another; all of the other patients harbored more than 100 parasites, and one patient harbored many more than 2000. The number of parasites could be estimated in only 11 cases.

Symptoms. This parasite is of unusual medical interest, as a number of those infected suffer from symptoms referable to its presence. In Ransom's[28] analysis of the cases reported up to 1904 the most frequent symptoms were of the nature of nervous or gastrointestinal disorders. The nervous symptoms ranged from mild disturbances, such as nervousness, irritability, and restlessness at

[25] Fourteen of these cases (I, II, III, IV, VI, VII, VIII, XIII, XIV, XVI, XVII, XVIII, XIX, XX) have been published in the February number of the Archives of Pediatrics. In this paper the parasite and ova are described and the recent literature reviewed. Three other cases (V, IX, and XII) will be reported in the Jour. Amer. Med. Assoc., April, 1910.

[26] New York Med. Jour., 1903, lxxviii, 877.

[27] Bull. 18, Hyg. Lab. U. S. Pub. Health and Mar. Hosp. Serv., 1904.

[28] Ibid.

night, to severer manifestations, such as choreiform movements and definite convulsive seizures.

Among the symptoms referable to the gastro-intestinal tract, pain or paresthesia are common. The pain is colicky, is usually referred to the epigastrium, and occurs in paroxysms. These attacks of pain may be infrequent, but they often occur several times a day. Ransom[29] refers to one case in which there was epigastric tenderness in association with the pain. Abdominal paresthesia, in the nature of a sudden sinking sensation, or a sudden feeling of "goneness," is not uncommon in older children. Diarrhœa sometimes occurs. Deaderick[30] has reported 6 cases of dwarf tapeworm infection, all of which presented symptoms which were attributed to the presence of the parasite. According to him the most common symptoms were, in the order of frequency, nausea, vomiting, œdema, headache, abdominal pain, diarrhœa, dyspnœa, and convulsions.

In 8 of my cases there were no symptoms attributable to the dwarf tapeworm infection; in 12 cases there were well-marked symptoms, which disappeared or were greatly ameliorated after appropriate treatment. The symptoms were mild in 5 cases, moderate in 4, and rather severe in 3 cases.

The most common nervous symptoms were restlessness at night, night cries, grinding the teeth during the night, and general irritability. Numbness and tingling in one hand was a symptom in 1 case. One patient (IX) had three general convulsions, and the disappearance of all symptoms since the expulsion of H. nana indicates that the intestinal parasites were the exciting cause. Itching of the nose and genital region were not infrequent.

The most common gastro-intestinal symptom was epigastric pain. This symptom was present in 7 cases. The pain was colicky, and was most often mild and transient, but in 2 cases it was quite severe. There were no abdominal signs in these cases; tenderness on palpation, muscle spasm, or rigidity were never present. Diarrhœa was present in 1 case, attacks of nausea and vomiting in 1 case, and nausea unaccompanied by vomiting in 1 case. The appetite was increased in 3 cases, capricious in 1, decreased in 5, and apparently normal in the remaining 4 cases. One patient complained of a sudden sinking sensation, referable to the abdominal organs.

Pain in the lower limbs was complained of in 2 cases, and in 1 it was quite severe. Œdema of the lower eyelids was a sign in 2 cases. Loss of weight was a rather prominent feature in 3 cases; in the other cases, however, even in those with severe symptoms, emaciation was not evident.

[29] Bull. 18, Hyg., Lab. U. S. Pub. Health and Mar. Hosp. Serv., 1904.
[30] Internat. Clinics, 1909, iv; Jour. Amer. Med. Assoc., 1906, xlviii, 2087.

TABLE IV.—HYMENOLEPIS NANA. CASES FROM 280 CONSECUTIVE EXAMINATIONS.

No.	Sex and age.	Nationality.	Parasites.	Symptoms.	Blood Examinations. Polynuclear neutrophile cells. Per cent.	Lymphocytes. Per cent.	Large mononuclear and transitional cells. Per cent.	Eosinophile cells. Per cent.	Basophile cells. Per cent.	Hemoglobin. Per cent.	Result.
1	Female 2	Russian parentage U. S.	....	None	28.4	28.9	39.0	2.4	—	80.0	About 200* parasites expelled by treatment. Feces free from ova sixteen days later. Second examination, twenty-six days later, also negative.
2	Female 2	U. S.	....	Restless at night, grinding of teeth and night cries one month. Irritable.	Leukocytes 15,000 per c.mm. Thoma–Zeiss apparatus 57.7	17.5	16.0	6.5	1.2	65.0	About 150* parasites expelled by treatment. Disappearance of symptoms. Feces free from ova sixteen and thirty-two days later.
3	Male 3	U. S.	....	Restless at night, grinding of teeth. Irritable. Frequent attacks of nausea and vomiting. Symptoms present six weeks	No apparent leukocytosis 42.5	41.5	8.0	6.9	0.5	70.0	About 100* parasites expelled. Eighteen days later very few ova still present in feces. Disappearance of symptoms.
4	Male 3	Italian parentage U. S.	....	None	No apparent leukocytosis 45.2	40.0	11.6	2.1	0.5	75.0	Feces free from ova eighteen days after treatment.
5	Male 3½	Italian parentage U. S.	....	Has not been entirely well for one year. Symptoms worse past month. Severe attacks of epigastric pain. Very restless at night. Night cries. Appetite very poor. Bowels constipated. Listless, pale. Puffiness of lower eyelids	No apparent leukocytosis Red blood cells 3,900,000 per c.mm. Thoma–Zeiss apparatus 26.8	45.6	16.0	10.9	0.2	55.0	Many more than 2000* worms expelled by treatment. Improvement continuous from time of treatment. No ova found in feces twenty days after treatment.
6	Male 4	Russian parentage U. S.	....	Restless at night, increased appetite. Frequent attacks of epigastric pain. Nasal pruritis	White blood cells 14,000 per c.mm. Thoma–Zeiss apparatus 60.0	18.0	12.8	9.0	0.1	70.0	Disappearance of symptoms after treatment. 150 to 200* parasites expelled. Very small number of ova in feces three weeks later.
7	Female 4	U. S.	....	For two months nocturnal eneuresis and genital and nasal pruritis. Appetite excessive, bowels normal	Leukocytes 16,000 Thoma–Zeiss apparatus 48.0	21.5	20.0	8.1	0.5	70.0	Disappearance of symptoms three weeks after treatment. Twenty days after treatment no ova found in feces. Eosinophiles 3.1 per cent.
8	Female 5	Italian parentage U. S.	....	Pale, frequent headaches, attacks of epigastric pain. Nervous, restless at night. Appetite poor, bowel movements normal	Leukocytes 13,000 per c.mm. Thoma–Zeiss apparatus 40.7	32.0	16.7	9.2	1.2	65.0	With exception of occasional headache is free from symptoms. Twenty days afrer treatment a very few ova were found in feces.

9	Male 5	U. S.	....	For two months, pale with loss of weight. Restless at night, night cries. Epigastric pain. Three general convulsions within past three weeks	Slight leukocytosis (estimated) 45.0 40.3 7.6 7.0 — 70.0	No ova found in feces twenty days after treatment. Marked and progressive improvement since treatment.
10	Male 5	U. S.	....	Frequent attacks of diarrhœa for about three months. Occasional attacks of epigastric pain. Restless at night, irritable. Appetite excessive	Slight leukocytosis (estimated) 48.8 26.9 10.4 11.0 1.2 75.0	Improvement progressiye after treatment. Feces contained no ova one month later.
11	Female 5	U. S.	....	No definite symptoms	No apparent leukocytosis 53.6 27.3 13.0 3.5 0.5 75.0	About 60* parasites expelled by treatment. No ova found in feces eighteen days later.
12	Male 6	U. S.	....	For one year poor appetite, pale, frequent attacks of epigastric pain. Child very listless. Constipated, poor appetite. Symptoms much worse for past month	No apparent leukocytosis 49.9 22.5 13.8 13.0 0.4 60.0	About 300* worms expelled by treatment. Relief of symptoms after treatment. Feces free from ova twenty and thirty-six days after treatment.
13	Male 6	Italian parentage U. S.	....	None	No apparent leukocytosis 66.0 20.3 11.0 1.1 0.5 70.0	Very small number of ova seen in feces three weeks after treatment.
14	Female 7	U. S.	....	For six months loss of weight. Pale, listless, easily tired. Frequent pains in lower extremities. Epigastric pain. Thin. Puffiness of lower eyelids. Appetite poor	Red blood cells, 3,200,000 per c.mm. White blood cells, 10,000 per c.mm. Normoblasts—microcytes Thomas-Zeiss apparatus 53.5 35.2 7.0 3.2 0.1 40.0	About 700* parasites expelled. Symptoms disappeared with exception of pains in limbs. Hb. 55 per cent. one month; 60 per cent. six weeks after treatment. Very small number of ova still found in feces.
15	Male 7	U. S.	....	None. Double infection, H. nana and T. dispar	No apparent leukocytosis 56.7 26.4 9.6 5.0 0.8 75.0	Very small number of ova in feces sixteen days after treatment.
16	Male 9	Italian parentage U. S.	....	Loss of weight five weeks. Easily tired, sleep disturbed. Night cries, grinding of teeth. Genital and nasal pruritis. Appetite capricious	Red blood cells, 3,800,000 per c.mm. White blood cells, 24,000 per c.mm. Thoma-Zeiss apparatus 44.3 20.2 11.1 22.6 1.2 65.0	Gain in weight, disappearance of symptoms after treatment. Few ova found in feces three weeks later. At this time eosinophile cells were 2 per cent.
17	Male 9	U. S.	....	No symptoms	No apparent leukocytosis 35.6 32.8 27.5 2.2 0.3 75.0	About 200* parasites expelled. No ova seen in feces three weeks after treatment.
18	Female 10	Russian parentage U. S.	....	Thin, nervous child. Nausea, sudden sensation of abdominal depression. Numbness and tingling in right hand. Pains in right thigh. Poor appetite. Loss of weight	Red blood cells, 4,100,000 per c.mm. Leukocytes, 16,000 per c.mm. Tnoma-Zeiss apparatus 50.7 21.2 20.5 7.2 0.2 60.0	About 600* parasites expelled by treatment. Disappearance of symptoms. No ova found in feces sixteen and thirty-two days later.
19	Female 10	Sicily	....	Rather thin. No definite symptoms. Double infection with H. nana and T. dispar	No apparent leukocytosis 42.2 40.8 14.8 2.7 — 70.0	Very small number of ova in feces sixteen days after treatment.
20	Female 11	U. S.	....	None	No apparent leukocytosis 42.0 31.2 22.5 4.0 0.3 80.0	About 50* parasites expelled. No ova found in feces sixteen days and one month after treatment.

* The numbers given represent the intact worms and parts consisting of nearly the whole worm. The parasites become broken up, and an exact count is impossible. The figures are probably too low. The number of parasites could be estimated in only 11 cases. In the other cases, none or only a small portion of the feces was preserved after treatment.

Blood Examinations. The blood examinations present points of especial interest. With a single exception, the percentage of eosinophile cells was increased in the patients who suffered from symptoms. The exceptional case was one of rather long standing infection with pronounced secondary anemia. The eosinophile cells were between 6 and 10 per cent. in 7 cases, between 10 and 20 per cent. in 3 cases, and above 20 per cent. in 1 case. The eosinophile cells were above 5 per cent. in only 1 of the cases without symptoms, and in this case the eosinophile cells were 5.2 per cent. The hemoglobin was determined in all cases, and in a number the percentage was below normal. The red blood cells were counted in 4 cases (V, XIV, XVI, and XVIII), and in all a secondary anemia was present. The degree of anemia may be characterized as mild in 1 case, moderate in 2, and rather severe in 1 case. In 1 case microcytes and normoblasts were present in the blood. In a number of the cases with eosinohpilia the number of leukocytes seemed to be increased.

A case of infection with Hymenolepis nana is cited by Bücklers,[31] in which the eosinophile cells were 7 per cent. No mention is made in this case of the presence or absence of symptoms. Deaderick[32] has reported 6 cases of dwarf tapeworm infection which showed an eosinophilia of 11.5, 15, 9, 26, 8.2, and 7.8 per cent., respectively. All of these patients had symptoms apparently due to the parasites.

Treatment. Oleoresin of male fern is the remedy generally recommended, and is quite effective. Before the administration of the anthelmintic it is desirable to have the intestinal canal as empty as possible. To accomplish this the diet should be restricted to easily digested food for several days and a cathartic given each day. On the evening before the specific treatment a cathartic should be given; the oleoresin of male fern should be administered the following morning. It is of importance that the remedy be fresh, and it is best administered on an empty stomach. The dose will naturally vary with the age of the patient; from ½ to 1 dram is sufficient, and has, in my experience, been entirely harmless. It is best to give the male fern in three to five doses, administered at half-hour intervals. One-half hour after the last dose of male fern a brisk saline purge should be given.

A single treatment is not always sufficient, but its effectiveness can be determined by a later examination of the feces for ova. When worms are left in the intestinal canal, or develop after treatment, the ova usually reappear after an interval of about fifteen days.

Ascaris lumbricoides (the common roundworm, the eelworm). Table V; Cases I and II, Table I. In the first group of examina-

[31] Münch. med. Woch., 1894, xli, 22 and 47.

[32] Jour. Amer. Med. Assoc., 1906, xlvii, 2087; Internat. Clinics, 1909, iv.

TABLE V.—ASCARIS LUMBRICOIDES. CASES FROM 280 CONSECUTIVE EXAMINATIONS.

No.	Sex and age.	Nationality.	Parasites.	Symptoms.	Blood Examinations. Polynuclear neutrophile Cells. Per cent.	Lymphocytes. Per cent.	Large mononuclear and transitional cells. Per cent.	Eosinophiles. Per cent.	Basophile cells. Per cent.	Hemoglobin. Per cent.	Result.
1	Male 4	U. S.	....	Irritable for six weeks. Nasal pruritis, poor appetite. Very restless at night. See No. 5, Chart II	46.2	32.0	10.5	9.0	1.2	65.0	Expulsion of one ascaris. One threadworm in feces after treatment. Still pale, relief of other symptoms.
2	Male 5	U. S.	....	Pale, restless, poor appetite. Occasional attackss of nausea and vomiting	No apparent leukocytosis						Expulsion of one roundworm. Relief of symptoms.
3	Male 5	U. S	....	Very nervous, restless at night, night cries. Pale, poor appetite. Two years ago was very ill. Jaundice at this time. Recovery (?) after expulsion of roundworm	50.5	31.0	9.6	6.5	0.5	70.0	
					No apparent leukocytosis						Improvement after expulsion of one roundworm.
					45.5	26.2	18.7	9.2	0.2	70.0	
4	Male C	U. S.	....	None	Slight leukocytosis (estimated)						Untreated. Lost track of.
5	Female 6	U. S.	....	None. See No. 16, Chart II	59.9	32.1	4.9	2.4	0.4	80.0	
					42.0	40.2	14.3	1.8	0.5	70.0	Expulsion of one roundworm. Two threadworms found in feces after treatment.
6	Female 7	Italian parentage U. S.	....	Pale, poor appetite, restless at night	Slight leukocytosis (estimated)						Expulsion of one roundworm. Improvement.
					64.2	22.0	6.1	6.9	—	70.0	

tions (Table I) there were 2 cases of infection with this parasite; there were 6 cases (2.14 per cent.) in the 280 consecutive examinations.

The 2 cases from the first group are of sufficient interest to give in some detail. In the first case, the onset of the illness was sudden, with vomiting and loss of appetite. The child was very restless at night, and would frequently awake and cry out as if in pain. For ten days these symptoms continued; the evening temperature ranged from 101° to 102° F., and the respirations and pulse were proportionately increased. The patient was thoroughly examined, but nothing could be found to account for the symptoms. On the tenth day of the illness a blood count showed the presence of an eosinophilia (12.1 per cent.). This led to an examination of the stools, and the ova of ascaris were found in great numbers. The expulsion of a lumbricoid worm by santonin was followed by disappearance of all symptoms. An almost identical case is cited by Still.[33]

The symptoms in the other case (Table II), were less acute. For three months the patient had been irritable, pale, and had lost weight. At the time of admission he had an attack of jaundice; the skin, sclerotics, and mucous membranes were distinctly yellow. The stools were colorless, and the urine contained bile pigments. At this time the eosinophile cells were 12.6 per cent., and the ova of A. lumbricoides were found in the feces. After treatment, two ascarides were expelled. The jaundice lasted for ten days; after its disappearance the other symptoms improved, and the child began to gain in weight. This patient had passed a roundworm one month previous to the onset of the recorded illness.

Two of the 6 cases from the consecutive examinations suffered from no symptoms referable to helminthiasis; 3 of the remaining 4 cases suffered from mild symptoms; in one case the symptoms were rather severe. The more pronounced symptoms in these cases were the ones commonly due to the presence of this worm: loss of color and weight, poor appetite, restlessness at night, and night cries. One patient (II) suffered from attacks of nausea and vomiting, which have not recurred since the expulsion of one lumbricoid worm.

The past history of Case III was rather interesting. Two years before admission the child had an acute illness, accompanied by fever and jaundice. According to the mother's story, recovery ensued immediately after the passage of one lumbricoid worm.

Blood Examinations. In the 2 cases without symptoms the eosinophile cells were not increased. In all of the cases with symptoms, including the 2 cases from the first group of examinations, there was a moderate degree of eosinophilia. The percentages of eosinophile cells varied from 6.2 to 12.6.

[33] Common Disorders of Childhood, 1909.

Treatment. with santonin is effective, the details of which are given in all of the text-books. Experiments have shown that it takes about one month for the development of this worm from the ovum to the sexually mature parasite. Therefore, in order to be sure of the thoroughness of the treatment, the feces should be examined for ova after one month.

Tenia saginata (T. mediocanellata, the fat, or beef tapeworm). Table VI. This parasite was found in 5 cases (1.74 per cent.), and in 3 cases the segments had been seen by the mother, who was consequently aware of the infection. In 2 cases (II and III) the diagnosis was made by finding the ova in the feces, and later confirmed by the discovery of the segments.

Symptomatology. Two of the 5 patients suffered from no symptoms referable to the tapeworm. Two of the remaining 3 patients suffered from nervousness; 1 child had become quite irritable, and 1 was very restless during sleep. The appetite was at times excessive in 1 case; in 2 cases it was meagre. One patient suffered from frequent attacks of abdominal colic, and the pain was referred to the epigastrium. None of the patients showed signs of emaciation.

Blood Examinations. The blood was not examined in the 2 cases without symptoms. In the 3 patients who suffered from symptoms the percentage of the eosinophile cells was increased, and ranged from 7.1 per cent. to 13.2 per cent.

Treatment. The treatment followed in these cases was that given under Hymenolepis nana.

General Discussion. *Symptomatology and Pathology.* The obscurity of the symptoms of helminthiasis and the irregularity with which they occur has led to uncertainty and confusion. It is well known that in many instances intestinal worms produce no appreciable effect, while in other cases they may be responsible for definite symptoms which are always deleterious and sometimes severe.

The occurrence of symptoms in infection with the common intestinal worms seems to be, to some extent, dependent on the number of the parasites present. This factor, however, can be of little importance with parasites, of which, as a rule, only a single worm or a small number of worms are harbored. The cases collected by Becker,[34] in which the whipworm was responsible for severe symptoms, were all infected with large numbers of the parasite. In my cases it was in instances in which many threadworms were being passed that the symptoms were most marked. As a rule, the cases of dwarf tapeworm infection which harbored the largest number of worms suffered the most noticeable effects. On the other hand, one finds numerous references in the literature which

[34] Deut. med. Woch, June 26, 1902, 648.

TABLE VI.—TENIA SAGINATA. CASES FROM 280 CONSECUTIVE EXAMINATIONS.

No.	Sex and age.	Nationality.	Parasites.	Symptoms.	Blood Examinations. Polynuclear cells. Per cent.	Lymphocytes. Per cent.	Large mononuclear and transitional cells. Per cent.	Eosinophiles. Per cent.	Basophile cells. Per cent.	Hemoglobin. Per cent.	Result.
1	Female 6	U. S.	....	Very nervous and irritable. Appetite at times excessive. Good color, well nourished. Has passed segments for two years	Slight leukocytosis (estimated) 44.0	16.1	25.2	13.2	0.7	80.0	Expulsion of tapeworm. Head not found. Segments had not reappeared four months later. Freedom from symptoms.
2	Male 9	U. S.	....	No symptoms referable to tapeworm. Mother not aware of infection	Not made						Did not return for treatment.
3	Male 10	U. S.	....	No definite symptoms. Child pale, poorly nourished. Mother not aware of infection	Not made						Expulsion of tapeworm. Head not found. Not seen since two days after treatment.
4	Female 10	U. S.	....	Pale. Restless at night. Appetite poor. Has passed segments for one year. Child is very nervous	Moderate leukocytosis (estimated) 48.3	18.5	23.6	9.0	0.3	55.0	Expulsion of entire worm. Improvement.
5	Male 12	Armenian	....	Pale, poor appetite, frequent attacks of colic. Pain referred to epigastrium. Segments observed by mother for eighteen months	No apparent leukocytosis. 42.0	20.0	30.0	7.1	0.4	70.0	Expulsion of tapeworm. Head not found. Patient not seen since two weeks after treatment. Improvement.

show that large numbers of parasites may be harbored with no apparent discomfort to the host. Dehio[35] believes that "Bothriocephalus anemia" is only produced after the death or disease of the parasite, but even if this is true, it seems scarcely possible that this factor comes into play with the common helminth.

The age of the patient is of great importance, for it is well known that reflex nervous disturbances are more likely to occur in children than in adults. The species of parasite is of some importance, but in infections with all of the parasites considered in this paper symptoms are inconstant. There is no adequate explanation for this irregularity in the occurrence of symptoms, but it may be due to a definite predisposition on the part of some individuals, or the worms may excrete toxic substances only under certain conditions, of the nature of which we are ignorant. It is not improbable that both factors are of importance.

As to the ultimate cause of the symptoms of helminthiasis there is little positive knowledge and much speculation. There have been experiments which indicate that some parasites at least excrete toxic substances which may have an influence on the host, and clinical experience often lends support to this view. A case has recently been reported by Artaega[36] in which ascarides were the probable cause of profound hemolysis. On the other hand, there are symptoms the nature of which suggests an irritative action, such as diarrhœa and the local irritation produced by migrating oxyurides. Many of the nervous disturbances are probably reflex, due to the irritation set up by the worms.

It has been mentioned that the intestinal canal of the whipworm was found to contain blood pigment,[37] and that the feces of those infected with this parasite reacted to an occult blood test.[38] Autopsies have shown that the head of the dwarf tapeworm burroughs into the intestinal mucosa, and that considerable inflammation may be thus produced. Without undue speculation, it can be safely said that the present state of our knowledge indicates that the influence of the common intestinal worms is due to direct irritation, to the abstraction of blood, or to toxic substances excreted by the parasite.[39]

[35] Quoted by Emerson, Clinical Diagnosis, 1906, p. 389.

[36] Abst. Jour. Amer. Med. Assoc., May 8, 1909.

[37] Deut. Archiv f. klin. Med., 1896, lvii, 104.

[38] Semaine méd., xxix, p. 35.

[39] No mention is made of the well-known mechanical effects due to the migration of Ascarides. The various means by which parasitic worms may produce injury is summarized by Stiles as follows: (1) Nourishment is taken which should go to the host; (2) blood is taken by the parasites as food; (3) mechanical pressure irritates or causes atrophy of organs or parts of organs; (4) natural channels may be obstructed; (5) the wandering of the parasites may cause irritation; (6) substances may be excreted which may have a toxic influence, and which may change the natural condition of the body fluids (blood); (7) injury to the intestinal mucosa or to the skin may form points of entry for bacterial or protozoan infections (Osler and McCrae, Modern Medicine, vol. i, 1907).

The most important symptoms of helminthiasis may be tabulated as follows:

I. Gastro-intestinal symptoms.
- (*A*) Nausea.
- (*B*) Vomiting.
- (*C*) Diarrhœa.
- (*D*) Abdominal pain.
- (*E*) Jaundice. Commonest in ascaris infections. May be due to duodenitis or to mechanical obstruction of a bile duct by a parasite. May occur in tapeworm infections.
- (*F*) Abdominal paresthesia.[40]
  Sinking sensations, feeling of emptiness, sensation of "goneness," etc. Commonest in tapeworm infections.
- (*G*) Disturbances of appetite.
  1. Increased appetite.
  2. Decreased appetite.
  3. Capricious appetite.
  4. Perverted appetite.
- (*H*) Intestinal obstruction. (Ascarides.)

II. Symptoms of nervous organs. (May be reflex or tonic?)
- (*A*) Disturbances of sleep.
  1. Restlessness.
  2. Grinding of teeth.
  3. Night cries.
- (*B*) Irritability, nervousness.
- (*C*) Nasal pruritus.[41]
- (*D*) Dyspnœa.
- (*E*) Dizziness and vertigo.
- (*F*) Choreiform movements.
- (*G*) Convulsions.
- (*H*) Paralysis. (Functional.)

III. Symptoms referable to organs of special sense.
- (*A*) Perversions of—
  1. Sight.
  2. Hearing.
  3. Taste.
  4. Smell.
- (*B*) Pupillary changes.

[40] This is really a nervous symptom, but since the sensations are referred to the abdomen, it is placed in the above heading..

[41] The origin of this symptom is obscure. Its relationship to helminthiasis is doubted, probably because many children not harboring intestinal worms have the habit of picking or scratching the nose. Nasal itching, however, is mentioned by most authorities on helminthiasis, and appears in the statistics of Cobbold and Hirsch, on the symptoms of tapeworm infections.

IV. Symptoms referable to the skin or due to irritation of the skin or mucous membranes.
  (*A*) Symptoms referable to skin.
    1. Erythema. } Ascaris, tapeworms.
    2. Urticaria. }
  (*B*) Rectal irritation (Oxyuris vermicularis).
  (*C*) Genital pruritus,[42] or irritation, which may lead to—
    1. Vulvitis or vulvovaginitis. } Usually in infections
    2. Enuresis. } with Oxyuris ver-
    3. Masturbation. } micularis.
V. General symptoms.
  (*A*) Loss of weight.
  (*B*) Anemia.

Blood Changes. Müller and Reider,[43] in 1891, and Zappert,[44] in 1893, found an increase of the eosinophile cells in cases of uncinariasis. Following these observations, eosinophilia has been noted in infections with many varieties of parasitic worms. In the case of the more common and often harmless parasites the recorded observations show that eosinophilia may occur, but is inconstant.

In the blood counts made by Boycott[45] in cases of oxyuris infection, about two-fifths of the cases showed an eosinophilia. This inconstancy has been noted by other observers.

From his studies on uncinariasis, Boycott[46] is of the opinion that the presence and degree of eosinophilia is in inverse proportion to the duration of infection. He found in cases of hookworm infection that the eosinophilia gradually disappeared without the worm leaving the intestine. Ashford and King,[47] in their work on uncinariasis, found that there was no increase in the eosinophile cells in severe infections or in those of long standing associated with anemia.

These clinical observations find confirmation in the experimental work of Opie[48] on trichinosis. This investigator administered estimated numbers of the encysted embryos of Trichina spiralis to guinea-pigs, and observed the effect of the infection on the eosinophile cells. He found that the administration of a moderate number of trichinæ produced eosinophilia, but when a severe infection was induced, the eosinophile cells decreased or disappeared, and death of the animal ensued.

[42] This symptom is probably not always due to local irritation, since it may occur in tapeworm infections. With the larger tapeworms the passage of segments may be the causative factor. This could hardly explain its occurrence in the case of the dwarf tapeworm, where the segments are extremely small and do not seem to be regularly passed.

[43] Deut. Archiv f. klin. Med., 1891, xlviii, 96.

[44] Zeitschr. f. klin. Med., 1893, xxiii, 227.

[45] Brit. Med. Jour., 1903, ii, 1267.

[46] Jour. Hyg., 1903, iii, 95; 1904, iv, 437.

[47] Amer. Med., 1903, vii, 391.

[48] Amer. Jour. Med. Sci., 1904, cxxvii, 477.

From the blood counts in my cases it seems that the occurrence of eosinophilia bears relation to the presence of symptoms and to the duration of infection. In other words, eosinophilia was generally absent in cases which presented no symptoms of helminthiasis (usually, but not always, light infections). Eosinophilia was usually present in cases which presented symptoms, with the exception of severe or long-standing infections. The degree of eosinophilia did not seem to bear any constant relation to the severity of the symptoms.

The above statements are not strictly applicable to the whipworm, since this parasite did not cause an increase of the eosinophile cells. On the other hand, it rarely causes symptoms. The significance of the percentages of the eosinophile cells found in children who harbor intestinal worms is, obviously, dependent on the percentages of these cells found in normal children.

It is often stated that in children the normal percentages of the eosinophile cells are much greater than those considered normal for adults. The investigations of Carstanjen[49] on children do not show that the percentage of these cells is uniformly high. They indicate, however, that the percentages may vary greatly in children of the same age. Thus, in children between four and five years of age, the eosinophile cells in one case were 0.75 per cent., in another 16.65 per cent. The eosinophiles were above 6 per cent. in 16 of the 55 children between two and thirteen years of age. The counts of Zappert[50] have practically the same significance; 16 of the 28 children between two and thirteen years of age showed an eosinophilia of more than 6 per cent. In one case, a child with chorea, the eosinophiles were 19.54 per cent. As shown in the reports of these writers, a number of the children suffered from various chronic disorders, and therefore cannot be considered entirely normal. The attempt to exclude helminthiasis is not mentioned in any of these investigations, and apparently was not made.

Boycott[51] found the eosinophile cells under 5 per cent. in 8 out of 10 normal and apparently "wormless" children. In one case the eosinophile cells were 5.2 per cent.; in another, 5.4 per cent. I have made differential blood counts on 20 apparently normal children who did not harbor intestinal worms—judging from an examination of the feces (Table VII); 14 of these children appeared normal, and complained of no symptoms; 6 were recovering from mild digestive disorders. In 18 cases the eosinophile cells were below 5 per cent., in 1 case they were 5 per cent, and in 1 case 6 per cent.

The possibility of an idiopathic eosinophilia in children cannot

[49] Jahr. f. Kinderheilk, 1900, lii, 215, 233, and 684.
[50] Ztschr. f. klin. Med., 1893, xxiii, 227.
[51] Brit. Med. Jour., 1903, ii, 1267.

be excluded on the basis of this small number of examinations. The results, however, are of sufficient uniformity to indicate that in normal children—not harboring intestinal worms—the eosinophile cells are not frequently above 5 per cent. This question, however, is worthy of further study.

There have been a number of experiments which throw light on the cause of eosinophilia in infections with parasitic worms. Accumulation of eosinophile cells in the intestinal wall has been observed by Strong[52] and Yates[53] in postmortem examinations of fatal cases of uncinariasis. A local accumulation of eosinophile cells in the muscles containing encysted trichinæ has been observed by Brown,[54] Opie,[55] and others. Calamada[56] was able to produce eosinophilia in rabbits and guinea-pigs by the injection of a filtered aqueous extract of Tenia saginata.

Table VII.—The Percentage of Eosinophile Cells in Apparently Normal Children.

No.	Age Years.	Per cent. of eosinophile cells.
1*	2	5.0
2†	2	2.8
3†	2	2.3
4†	3	3.9
5*	3	6.0
6†	3	4.3
7*	5	3.2
8†	5	0.9
9†	5	3.8
10*	5	2.1
11†	5	4.7
12†	6	1.2
13†	6	0.8
14*	6	0.01
15*	8	3.7
16†	8	4.0
17†	8	0.2
18†	10	1.6
19†	10	2.4
20†	11	3.5

* Patients from private practice. † Dispensary patients.

These experiments indicate that the parasites probably excrete substances which have a positive chemotactic influence on the eosinophile cells. Moreover, it is probable that the eosinophilia represents a reaction on the part of the organism, and that in severe or long-standing infections the power of producing eosinophile cells is gradually diminished.

The association of Charcot-Leyden crystals with eosinophilia has been observed in several diseases. These crystals frequently occur in the feces in helminthiasis, and their presence is of considerable

[52] Quoted by Opie.
[53] Johns Hopkins Hosp. Bull., 1901, xii, 366.
[54] Jour. Exper. Med., 1898, iii, 315.
[55] Amer. Jour. Med. Sci., 1904, cxxvii, 477.
[56] Cent. f. Bakt. u. Parasit., 1901, xxx, 375.

diagnostic value. Charcot-Leyden crystals are probably derived from the eosinophile cells, so that their occurrence in the feces in helminthiasis would seem to represent a local eosinophilia.[57]

Bücklers[58] has noted the presence of Charcot-Leyden crystals in the feces of cases of helminthiasis showing eosinophilia. I examined the feces for Charcot-Leyden crystals in 14 cases with eosinophilia, and the result was positive in the following: in 1 of 6 cases infected with H. nana, in 4 of 6 cases infected with O. vermicularis, and in both of 2 cases infected with T. saginata.

In a number of my cases the large mononuclear and transitional cells were above the percentages usually given as normal. The apparent increase in these cells seemed of no especial significance, and had no relation to the presence of symptoms or to the severity of the infection. The percentage of basophile cells (mast cells) was frequently increased, and the increase was greatest in cases showing eosinophilia. This relationship, however, was not constant, and the mast cells were increased in several cases not showing eosinophilia.

The percentage of hemoglobin was low in many of the patients who suffered from symptoms. The anemia was more pronounced in the threadworm and dwarf tapeworm infections.

Diagnosis. It seems hardly necessary to state that it is impossible to diagnosticate the presence of intestinal worms from the symptoms produced in the host. The symptoms of helminthiasis are usually obscure and are more often due to other causes.

The presence of Tenia saginata is usually indicated by the passage of segments, but, as previously shown, these may not be observed. The migration of oxyurides and the local symptoms produced often leads to their detection. The presence of ascarides may be indicated by the previous passage of a worm. Segments of the dwarf tapeworm are occasionally found in the stools, but they are so minute that they can only be recognized by means of a lens. Rarely the intact worms may be passed after the administration of a cathartic. The whipworm is rarely, if ever, found in the stools.

The easiest and only satisfactory diagnostic method is the examination of the feces for the parasites, their parts, or ova. It is best to administer a calomel purge before obtaining the specimen for examination, as by this means oxyuris is more likely to be detected. A number of methods of examination have been recommended, but I have found the following to be entirely satisfactory: A small portion of the feces (15 to 20 grams) is thoroughly mixed with sufficient distilled water to make a translucent mixture. This is well shaken, and a large drop is placed on a slide and covered with a cover-slip. By means of the mechanical stage eight to ten preparations are thoroughly examined. Two by three inch slides and

[57] Limasset, Thèse de Paris, 1901. [58] Münch. med. Woch., 1894, xli, 22 and 47.

one by two inch cover-glasses are more convenient than the ordinary size, as more material can be examined in a single specimen. In conducting the microscopic examination, a moderate illumination is desirable, and it is best not to use a condenser. Transparent and colorless ova, such as those of the dwarf tapeworm, the threadworm, and hookworm, are likely to be overlooked if the illumination is too intense.

It is rather important that the feces be thoroughly mixed, since the ova of parasites inhabiting the upper intestinal tract are more likely to be found in the centre of the fecal mass, while the ova of other parasites are only discharged in the large intestine, and in consequence are more likely to be in the external layer. Scrapings from the rectum frequently give positive results with the threadworm when the examination of the feces is negative.

One finds rather frequent references to pseudo-ova, which may lead to confusion. These bodies are usually vegetable cells, which have a cell membrane and cellular or granular contents. After the ingestion of some of the common fruits—oranges, raspberries, bananas, etc.—these cells are frequently found in the feces. Starch granules and epithelial cells may occasionally have a superficial resemblance to ova. Although these bodies may be a source of confusion, yet their resemblance to true ova is only superficial. All danger of confusion is eliminated by familiarity with the appearance of the ova of intestinal worms. Absolute verification of the diagnosis may be obtained by recovery of the parasite.

To search for parasites the feces should be well diluted and poured into a flat vesesl, the bottom of which has been painted black. By this means the worms can be easily recognized, and their species determined by microscopic examination.

If further argument were needed to show the importance of examining the feces for ova, it is only necessary to recall my statistics. The parasites which rank first (the whipworm) and third (the dwarf tapeworm) in frequency are never observed by the patients, but can only be detected by finding the ova on microscopic examination of the feces.

Prophylaxis. With the exception of Tenia saginata, infection with the parasites dealt with in this paper results from the ingestion of ova. Infection with Tenia saginata occurs from ingestion of so-called "measley beef," that is, meat containing the cysticercus stage of this parasite. The tongue and muscles of mastication most often contain the cysticerci. The exclusion of all infected meat by rigid inspection is the best preventive measure. Heat destroys the embryos, and thorough cooking of infected meat will render it harmless. To prevent infection with the other parasites, it is important that infected cases should be thoroughly and promptly treated. Measures should be taken to prevent contamination of the water supply with the ova. To prevent infection of other

members of the family, rigid cleanliness should be observed, and the contamination of food or hands with the feces of infected persons guarded against. An experiment of Stiles[59] indicates that flies may be the carriers of the ova of the eelworm of hogs—a parasite closely related to the eelworm of man. It is possible that infection with the human parasite may be disseminated in this manner. The prophylaxis is obvious. It is possible that infection with the dwarf tapeworm may result from contamination of food with the feces of rats or mice infected with this worm. This gives another indication for the extermination of rats and mice. To prevent the spread of intestinal worms, it is only necessary to remember that the feces of those infected usually contain great numbers of the ova, and that the ingestion of a single ovum may lead to the development of an intestinal worm.

Summary. 1. Twelve of 30 children who suffered from unexplained nervous or gastro-intestinal symptoms were found to harbor intestinal worms.

2. Consecutive examinations of 280 children showed that 80 (28.57 per cent.) harbored intestinal worms. Five of the children harbored two species of parasite, giving a total of 85 infections.

3. Thirty-one (11.07 per cent.) of the children harbored Trichuris trichiura, 23 (8.21 per cent.) harbored Oxyuris vermicularis, 20 (7.14 per cent.) harbored Hymenolepis nana, 6 (2.14 per cent.) were infected with Ascaris lumbricoides, and 5 (1.78 per cent.) with Tenia saginata.

4. Only 1 of 33 children infected with Trichuris trichiura (from both groups of examinations) suffered from symptoms.

5. Thirty-five of the 51 children infected with the other parasites (from the consecutive examinations) suffered from symptoms.

6. The eosinophile blood cells were not increased in cases infected with Trichuris trichiura.

6. In infections with the other parasites eosinophilia was usually absent when there were no symptoms due to helminthiasis. Eosinophilia was generally present in cases which presented symptoms of helminthiasis.

Conclusions. 1. Intestinal parasites are not infrequent among the children of the poorer classes of New York City.

2. Intestinal worms may be harbored without inconvenience to the host. On the other hand, symptoms may occur which are always deleterious, and sometimes severe.

These investigations were made on patients from the clinic of Dr. Thomas S. Southworth at the out-patient department of the Babies' Hospital, and from the service of Dr. Eli Long, at the New York University and Bellevue Hospital Medical College. I desire to express my appreciation for this privilege and for encouragement

[59] Modern Medicine, Osler and McCrae, 1907, i, 597.

in carrying out the work. I wish to acknowledge my indebtedness to Miss Eleanor Ketcham, visiting nurse to the children's clinic of the New York University and Bellevue Hospital Medical College, for valuable assistance in obtaining material and in following the cases.

AN EPIDEMIC OF NOMA.

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Through the kindness of Dr. Jonas E. Reinthaler and Dr. Charles A. Elsberg, respectively attending physician and surgeon to the New York Hebrew Infant Asylum, I had the opportunity of studying an epidemic of noma which occurred in that institution in the spring of 1909. The asylum is a substantial, well-preserved edifice. It was originally a private dwelling, and was later enlarged by the addition of wings. The institution accommodates about 150 children. During the epidemic there were 140 children in the asylum of varying ages, up to six years.

Three cases of noma occurred in the asylum during fourteen years, one in November, 1902, one in February, 1907, and one in February, 1908. All three patients died; in two the treatment was conservative; in the third and last a wide excision was performed by Dr. Elsberg. In the three cases the disease developed in the course of epidemics of measles complicated by ulcerative stomatitis. It is of interest that the three cases, though widely separated in time, occurred in the same ward—a sunny, well-ventilated room.

The epidemic of noma of 1909 complicated an epidemic of measles, which spread in the institution despite careful isolation of all the exposed children. The epidemic of measles was a severe one and complications were frequent. There were 81 cases—of which 13 developed diphtheria, and 24 pneumonia—with a mortality (exclusive of noma) of 4 per cent. Although special attention was paid to the mouths, ulcerative stomatitis occurred in fully 25 per cent. of the children, and it was among these children that most of the cases of noma developed. There were 8 cases of true noma and 3 doubtful ones, to which reference will later be made.

One of the most striking facts about noma is that it has formerly broken out, almost invariably, in overcrowded and ill-regulated hospitals. For example, Saviard and Poupart[1] recorded epidemics of noma in the old Hôtel Dieu in Paris under such circumstances; in the new institution the disease did not develop. There are many similar reports in the literature of the disease. On the other hand,

[1] Trans. Med. Chir. Soc., Edinburgh, 1892–9, xii, 251.

in recent years epidemics have broken out in excellent institutions, as in that reported by Blumer and McParlane[2] and in that of Crandon, Place, and Brown.[3]

The association of noma with the infectious diseases—especially measles and pertussis—is well known. The pronounced tendency for noma to appear in the spring and fall may depend upon the greater frequency of the infectious diseases in those seasons. There is no conclusive proof that the disease is contagious. Mayr[4] believes in its contagiousness; Holt[5] is of the same opinion, as he has seen 5 cases of noma, after whooping cough, develop in the same ward. Of the cases in our epidemic, 2 developed in one ward, 3 appeared in another, and 5 (including the three not proved) developed in a third ward, which was reserved, as far as possible, for all of the cases of stomatitis. A single case developed in the isolation house to which the child, suffering from diphtheria, had been transferred. Two of the wards were on the same floor, the third was on another. Each ward was carefully isolated, had its special nurses, separate food, dishes, etc. The children who developed noma were transferred to the isolation house as soon as the disease appeared. It is often impossible to say when noma is developing, and undoubtedly many children were exposed to the disease when they were presumably in a receptive condition--suffering from measles and ulcerative stomatitis. With these conditions, favorable for the spread of a contagious disease, only a few of the little patients were attacked by noma, and no connection could be demonstrated between the isolated cases.[6]

Noma may appear at any age and among all cases of patients. The large majority of the cases have occurred among poorly nourished children[7] during the first and second dentition, and many apparently developed from ulcers around the teeth. Rilliet and Barthez,[8] in their classic description of the disease, describe noma in infants at the breast. Inasmuch as noma so often follows ulcerative stomatitis, many writers (Eichhorst,[9] Henoch,[10] Guizetti,[11] Seiffert,[12] etc.) consider noma an advanced stage of stomatitis. They point out that at first it is a purely local disease, that the same organisms

[2] Amer. Jour. Med. Sci., 1901, cxxii.

[3] Boston Med. and Surg. Jour., April 15, 1909.

[4] Zeitschr. der Kais.-Kön. Gesellsch. der Aerzte zu Wien, 1852.

[5] Diseases of Infancy and Childhood, 1905, pp. 290, 692.

[6] Schmorl (Zeitschr. f. Thiermed., 1891, p. 375) described an epidemic, among rabbits, of gangrene beginning in the mouth, which was very similar to noma. He showed that it was contagious.

[7] Some of these cases in the literature described as noma in adults correspond very closely to cases of leukemia with terminal gangrenous ulceration in the mouth. Blood examinations were not recorded.

[8] Traité des Malad. des Enfants, 1852, p. 62.

[9] Specielle Patholog. und. Therap.

[10] Trans. Chicago Path. Soc., 1896, i, 252.

[11] Il Policlinico, 1899.

[12] Münch. med. Woch., 1901.

are found in the smears taken from the ulcers of both diseases, and that the transition from the one condition to the other has been observed. It will be shown below that a definite bacteriological picture is found in noma and not in ulcerative stomatitis.

There are usually no constitutional symptoms until the gangrene has begun to spread. The child is usually quiet and languid, but may be very restless and irritable. Often it is profoundly prostrated, but may feel well enough to sit up in bed and play, although the gangrene is spreading over its face. Marked pallor is an early symptom. The pulse soon becomes rapid and small; there is slight or no fever, and generally no pain. Thirst is marked, although the tongue is moist. Diarrhœa is often a serious symptom, but, according to some observers, occurs only when gangrenous material is swallowed.[13] Bronchopneumonia is the most frequent and fatal complication. It is of the aspiration type, and not uncommonly leads to abscess or gangrene of the lung.

Of the physical signs, the ulcer is usually the first lesion observed (Bohn,[14] Eichhorst, Osler,[15] etc.). Billroth, however, describes a nodule in the cheek as the starting point, and Fagge[16] believes the disease begins immediately under the mucous membrane. The necrotic ulcer becomes gangrenous, the adjoining portion of the cheek becomes intensely infiltrated, and the gangrene extends to it and often to the adjacent maxilla. The peculiarly penetrating foul odor from the mouth is at the beginning faint, yet it may be the first sign to call attention to the disease. As the overlying skin becomes involved, it assumes a violaceous hue, later turns black and is covered with vesicles; finally the gangrenous ulcer breaks through. Tourdes[17] describes three stages of the disease: (1) Ulceration of the mucous membrane, œdema of the face, infiltration of the cheek, lasting two or three days; (2) gangrene, lasting five to twelve days; and (3) the period of general infection. Rarely the disease runs a subacute course over several months. Finally, Gierke noted that noma may recur.[18]

In a large number of cases collected from the literature the mortality ranged from 70 to 100 per cent.

Many different kinds of treatment have been recommended.

[13] Several autopsies have shown a gangrenous condition of the gastro-intestinal tract. In 8 cases of noma of the genitalia and of the external ear observed by Gierke (Jahrb. f Kinderheilk., 1868, p. 65) diarrhœa was present in only one case, and then it was of short duration.

[14] Gerhardt's Handb. der Kinderk., iv.

[15] Practice of Medicine, 1905.

[16] Amer. System of Diseases of Children.

[17] Thèse de Strassbourg, 1848.

[18] In 20 cases he observed three recurrences—one four weeks after the discharge of necrotic tissue, a second case six months after the first attack, the third case after three years. Babes and Zambilovici (Annal. de l'Instit. de Path. et de Bact. de Bucarest, 1895, v) refer to several cases.

Some writers advise applications of alcohol or hydrogen peroxide, or potassium chlorate, nitric acid, etc. Others advise cauterization of the ulcer with the actual cautery; still others practise excision of the diseased area. No matter what the treatment employed, only isolated cases have recovered. It is important to make frequent cultures from the necrotic areas, as diphtheria may closely simulate true noma.[19] If there is any doubt as to the diagnosis, antitoxin should be given.

The bacteriology of noma rests on a definite basis since 1899, when Perthes[20] and Seiffert[21] independently described a bacterium or group of bacteria in this disease. Although, up to the present time, Koch's laws have not been fulfilled[22] the disease has not been experimentally reproduced and the bacteria have not been artificially cultivated—the constant presence of the bacteria in noma, and only in this disease, points very strongly to an etiological relationship. Perthes found that noma is due to a fungus-like growth belonging to the streptothrix group. At the border line between the gangrenous ulcer and normal tissue he found a thick branching network of fine fusiform threads—mycelium. From this mycelium single fine rods and spirilla extend into the normal tissue, surround the cells, and cause their death.[23] Krahn believes that the growth described by Perthes consists of two organisms—the spirillum sputigenum and spirochete dentium.[24] The majority of observers agree with Perthes and Seiffert. The same bacteriological picture was described in noma of other parts of the body by Matzenauer.[25] Perthes prepared his specimens for examination by treating the teased tissue or section from the edge of the ulcer—removed post

[19] Hektoen, in the discussion on Bishop and Ryan's paper before the Chicago Pathological Society, pointed out the close clinical correspondence between their cases of noma and cases of gangrene of the skin in which the Klebs-Loeffler bacillus is found. And Loeffler, at a meeting of the Greifswald Medizinische Verein, in 1890, called attention to the similarity between pathological specimens from cases of noma, shown by Grawitz (Deut. med. Woch., 1890)¶ and diphtheria in calves (Kälberdiphtherie) that he had observed.

[20] Arch. f. klin. Chir., 1899, lix.

[21] Münch. med. Woch., 1901.

[22] Hofman and Küster (Münch. med. Woch., 1904, 1907) found abscesses in animals after the bacteria were injected and found the same bacteria in the abscesses. Furthermore, they obtained (impure) anaërobic growths of the organism. Neither of these observations has been subsequently substantiated.

[23] Ranke (Jahrb. f. Kinderheilk., 1888, xxvii) and others have advanced evidence to show that the death of the cells is due to chemical influences.

[24] Miller (Microörganisms of the Human Mouth, 1890) has shown that these two organisms are normal inhabitants of the mouth, in small numbers. As all attempts at their cultivation have failed, he considers them parasites that cannot be separated from their hosts. In all forms of stomatitis, as well as in oral noma, these bacteria are present in enormous numbers in scrapings from the lesions. The fusiform "bacillus" of Vincent is also found on the surface of these ulcers. But it has not been demonstrated that any of these organisms are related to the streptothrix found in the tissues in noma, although some of the terminal filaments of the streptothrix resemble them closely (see Migula's (System der Bacterien, 1900) classification of these organisms).

[25] Archiv. f. Dermat. und Syph., 1902.

mortem—with dilute carbol-fuchsin for twenty-four hours and then briefly washing with alcohol.[26]

The clinical and bacteriological pictures of the cases of noma in our epidemic correspond in good part with the description given above. However, some features of importance in our cases warrant a description of them in some detail; the detail of the spread of gangrene, the appearance of the streptothrix in individual cases,[27] etc., will be omitted. By "conservative" treatment we mean topical applications of peroxide of hydrogen, pure alcohol, and potassium chlorate; by "radical" treatment, thorough cauterization of the ulcer and of adjoining tissue with the actual cautery.

CASE I.—*Measles; ulcerative stomatitis; noma of the vulva; recovery.*

Jennie W., aged two years, was always delicate. Bilateral chronic otitis media. Measles March 9, 1909, complicated by a severe ulcerative stomatitis. The latter cleared up except for one ulcer. This necrotic area became gangrenous in its centre. A specimen removed did not show the streptothrix of noma. On March 17 a grayish membrane was observed covering the vulva and extending over the labia minora and across the perineum to the rectum. Cultures for diphtheria were negative; antitoxin had no effect. The membrane spread, the affected area became necrotic in forty-eight hours, and there was a profuse discharge of a gangrenous odor from the vagina and rectum. A specimen removed from the edge of the ulcer showed the streptothrix of noma. After two weeks, during which period the child was profoundly prostrated, the discharge diminished and the ulcer assumed a healthier appearance. Although there was a considerable loss of tissue, little deformity remained when healing was complete. Treatment was conservative. During convalescence the ulcer in the mouth slowly healed.

CASE II.—*Measles, ulcerative stomatitis; diphtheria of the vulva; oral noma; recovery.*

Marie F., aged two and a half years; was always well and strong. On March 10 measles developed, in the course of which ulcerative stomatitis appeared. March 16, a membrane was first noticed on the vulva similar to that in the first case. Cultures showed Klebs-Loeffler bacilli; the membrane disappeared after antitoxin injections. As the ragged sloughing ulcers about the teeth showed no signs of

[26] Weaver and Tunnicliff (Jour. Infec. Dis., 1907) demonstrated that this streptothrix is decolorized by Gram's method. They obtained the best staining reactions by dropping n 10 per cent. saturated solution of alcoholic gentian violet in 5 per cent. phenol on the sectioa (that had been embedded in paraffin, treated with xylol, followed by absolute alcohol) for five minutes, clearing with aniline oil, washing with xylol, and mounting in balsam. A complete bibliography of noma is given by Weaver and Tunnicliff, Journal of Infectious Diseases, January, 1907.

[27] The streptothrix stained very well in our cases with the simple method of Perthes. I obtained the specimens by removing a small wedge of tissue from the edge of the ulcer, employing small straight scissors and forceps. A tonsillar "punch" may be employed to advantage.

healing, they were cauterized with the actual cautery on March 25. Following this all but one of the ulcers healed. The latter was situated near the right upper canine tooth; a section removed from it showed the streptothrix of noma. Treatment consisted of cauterization every second day. April 8, infiltration of the upper lip; ulcer much larger; superior maxilla exposed; overlying skin bluish. General condition good; slight fever and prostration. After fragments of the necrotic superior maxilla had separated, improvement began. The violaceous hue of the skin disappeared; the infiltration softened; and finally the slough separated from the ulcer. A specimen removed at this time showed numerous spindle-shaped rods extending into normal tissue, but no mycelium. The treatment was conservative from the time the gangrene began to spread.

Case III.—*Measles; diphtheria; oral noma; recovery.*

Isidor L., aged two years, had been previously well and strong. Measles March 18, complicated by faucial diphtheria. The membrane disappeared a few days after antitoxin injections. About one week later an ulcer appeared about the upper incisors. Despite several cauterizations, it spread until a large piece of necrotic maxilla was exposed. Treatment by cauterization was then stopped. A specimen removed showed the typical streptothrix. The upper lip became exceedingly firm and infiltrated, but the skin remained unchanged. During a period of ten days this condition was stationary; the child was listless and apathetic, he had no fever, pulse was rapid. Then a large fragment of necrotic maxilla became detached and was removed. Thereafter the local and general condition improved. Treatment was conservative after the spread of the ulcer. Little deformity remained after healing was complete.

Case IV.—*Measles; pneumonia; oral noma; death.*

Doris A., aged three years, was always pale and weakly. Enterocolitis in 1907, with recurrences from time to time. Measles March 8, complicated by a severe pneumonia. On March 17 an ulcer was seen on the mucous membrane of the right cheek. Cultures negative; antitoxin without effect; characteristic odor from the mouth. The cheek became indurated very rapidly, the overlying skin assumed the typical color, the ulcer spread to the vermilion border of the lip. The child died March 21, apparently overcome by toxemia. The treatment was conservative. A specimen was not removed.

Case V.—*Measles; diphtheria; ulcerative stomatitis; oral noma; death.*

Harry S., always well and strong. Measles March 12; tonsillar diphtheria March 19. The latter yielded to antitoxin injections. A mild form of stomatitis was present. On March 23 a ragged ulcer appeared below the lower central teeth, where there had been no previous lesion. A section showed the streptothrix of noma. Thor-

ough cauterization was practised; the next day the adjoining maxilla was exposed and the submaxillary region was infiltrated. On March 25 an ulcer appeared about the teeth of the upper jaw exactly opposite the gangrenous lesion of the lower—apparently a contact infection. It spread rapidly, and the eyelids and lip became puffy; an offensive discharge issued from the nostrils. The upper lesion spread more rapidly than the lower. Temperature ranged from 100° to 104°; pulse very rapid and small. The gangrenous ulcers finally perforated the skin over the chin and over the upper lip; pus appeared in the diarrhœal stool; the patient succumbed March 30.

CASE VI.—*Measles; pneumonia; ulcerative stomatitis; diphtheria; oral noma; death.*

Daniel B., aged three years; always ailing and on special diet for a year. Measles March 14; shortly after, a severe ulcerative stomatitis. Pneumonia with moderate prostration; convalescent by March 20. Faucial diphtheria on March 21, yielding to antitoxin injections. On March 23 an ulcer was first noted on the inner surface of the left cheek; at the same time a faintly gangrenous odor of the breath was observed. A section removed showed the lesion of noma. Although the ulcer was thoroughly cauterized, it spread and the overlying skin became necrotic; perforation occurred two days before death. The latter occurred on April 3 from a septic bronchopneumonia. This patient suffered considerable pain—an exceptional feature in our cases.

CASE VII.—*Measles; ulcerative stomatitis; oral noma; de th.*

Eddie A., aged two and a half years; was always strong and well-nourished. Measles appeared March 6, complicated by ulcerative stomatitis of moderate severity. On March 18 an ulcer was first seen in the normal mucous membrane along the frenum linguæ. With daily deep cauterization, this ulcer remained stationary, whereas the ulcers surrounding the teeth healed. A section taken from the sublingual lesion showed the streptothrix of noma; one removed from one of the other ulcers did not. On April 1 the ragged ulcer under the tongue began to spread on the surface and into the depths. It became gangrenous, spread over the whole floor of the mouth, and caused a marked induration in the submaxillary region. On April 4 an ulcer under the upper lip appeared, opposite the lower ulcer. It spread more rapidly than the original lesion. April 7, gangrene of the skin over both ulcers. Death on April 9.

CASE VIII.—*Measles; ulcerative stomatitis; oral noma; death.*

David R., aged two and a half years; always ailing and weakly. Had measles March 17, complicated by mild ulcerative stomatitis. March 22, apparently on the base of one of the ulcers about the lower central incisors there was a large deep ulcer. A specimen showed the streptothrix. The ulcer was frequently cauterized, and did not grow larger until April 4. It then began to spread, so that

by April 7 the chin was swollen and shiny. The next day a contact (?) lesion appeared on the upper gums and spread like the primary ulceration. A specimen removed showed the same pathological picture as that from the lower lesion. The skin of the chin became gangrenous, and soon after, gangrene of the upper lip developed. The patient died April 11; he was not prostrated until twenty-four hours before death.

In the last three cases frequent cauterization was employed after the ulcers had begun to spread, in order to determine its value in this stage of the disease. This radical treatment had no salutary effect on the lesion; if anything, it appeared to hasten the spread of the gangrene.

To these undoubted cases of noma I would add the three cases to which reference has already been made. All three patients had measles, and in two of them ulcerative stomatitis developed. In both the stomatitis cleared up with the exception of one ulcer. In the third case there was a single ulcer from the outset. The ulcer, in each case, was deep, ragged, and necrotic, identical with the ulcer observed in the pregangrenous stage of noma. Specimens taken from the edge of the ulcer showed in each case the streptothrix of Perthes. The ulcers were submitted to frequent and thorough cauterizations, and after a period of two to four weeks they healed without any spread of necrosis and without the development of serious constitutional symptoms.

Among the patients with stomatitis there were several who had very suspicious ulcers. Specimens removed did not show the streptothrix, and none of these patients developed noma.

It will have been noted that for the cauterizations and for the removal of specimens no anesthesia was employed. This was done for the following reasons: In the first place the manipulations may be carried out quite painlessly. In the second place, these children are already much weakened by their disease, and the function of their lungs is impaired by the associated pulmonary affection; in them a general anesthesia must certainly be very dangerous. In not a few of the patients who were operated upon under general anesthesia septic pneumonia followed.

In Cases II, VII, and VIII, the spread of gangrene was delayed about two weeks, in each case, by repeated cauterizations, and I believe that if cauterization had been begun earlier, the spread of noma might have been much longer delayed. It cannot, of course, be proved that the three cases of ulcer identical clinically and histologically with the pregangrenous ulcer of noma were really cases of noma aborted by radical treatment; the findings, however, all point in that direction. It appears to me, from our experience, that the radical treatment is of value only in the pregangrenous stage of noma. We have found the streptothrix in this stage, and hence have concluded that it is unnecessary to wait for the appearance of gan-

grene in order to institute radical treatment. As before stated, actual cauterization is as effectual as excision and is less mutilating.

The close association of noma with ulcerative stomatitis was seen in this epidemic. Ulcerative stomatitis of varying severity was present in 8 of the 11 patients; in some of the cases the ulcers were necrotic, almost gangrenous. Yet in only one (Case VIII) did it seem probable that noma had developed on one of the lesions of ulcerative stomatitis. From the observation of all the cases in this epidemic, and from the microscopic studies, we must conclude that disease of the mouth prepared a favorable soil for the development of noma, but that there was no evidence of a direct etiological connection between stomatitis and noma.

What significance should be attached, in Cases V, VII, and VIII, to the development of gangrenous ulcers—apparently by contact—cannot be determined. Such a path of transmission of the disease would seem probable; it occurred in three of our five fatal cases. In one of these patients the microscopic examination revealed the streptothrix in sections from the second ulcer. I have been unable to find any mention in the literature of second ulcers in noma.[28]

Conclusions. Noma usually appears in epidemic form; its contagiousness has not been proved. The disease is an entity, and not a later stage of ulcerative stomatitis; the latter offers a good soil for the development of noma. There is regularly present in noma a streptothrix characterized by a thick meshwork of mycelium at the border line between normal and necrotic tissue; fine rods and spirilla extend from mycelium into the adjacent tissues. The constant presence of this streptothrix, to the exclusion of other organisms, indicates that, in all probability, it stands in direct etiological relationship to noma. The streptothrix is present in noma before the disease is fully manifest—in the pregangrenous stage. It is in this stage of the disease that radical treatment is to be practised; after the ulcer spreads, the best results are obtained by conservative measures. General anesthesia should not be employed in any form of treatment because of the pronounced tendency to the development of septic pulmonary disease.

[28] Blood examinations were made in four cases. There was a marked anemia (2,000,000 red cells), with decided poikilocytosis and anisocytosis; no leukocytosis. The blood drop was very watery and was expressed with difficulty. Cells resembling myelocytes were present in the spreads. In three patients Wassermann tests were kindly made by Dr. Kaplan, bacteriologist to the Montefiore Home, with negative results. They were done because it has been suggested that the organism of noma is in the same class as Treponema pallidum.

THE ANTITRYPTIC ACTIVITY OF HUMAN BLOOD SERUM: ITS SIGNIFICANCE AND ITS DIAGNOSTIC VALUE.[1]

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It is not much more than a year ago that Brieger and Trebing announced that a new characteristic of the blood in cases of cancer had been determinend. This new feature consisted in a marked increase in the power of the serum to inhibit the proteolytic activity of solutions of trypsin, and was so constant an accompaniment of cancer as to be present in over 95 per cent. of the cases. Since the publication of their original paper, investigations on this subject have followed each other in rapid succession, so that the literature has come to assume a very goodly volume. As the result of this accumulation of data, it has become increasingly evident that the "antitryptic reaction," as it is called, is not an exclusive characteristic of cancer: it appears under certain other conditions of disease; it marks the change from breast to artificial feeding in infants; it is a striking feature of the onset of labor and the puerperium, as contrasted with pregnancy; in other words, it appears to be a physiological adaptation of widespread significance and value. On the other hand, the fact is not to be gainsaid that the accumulation of evidence has not materially weakened the assumption originally maintained by Brieger and Trebing—the antitryptic reaction is an almost constant accompaniment of cancer, and occurs in a very much smaller proportion of all other diseases. The significance of this association between cancer and this biological change in the character of the serum must be regarded as a matter of some importance, aside from any practical diagnostic application which may attach to it. It is part of the larger problem of the general constitutional influence exercised by the newgrowth upon its host. In a larger view, the physiological value of the reaction, and its general relation to the subject of immunity, is a matter which requires elucidation. In the present paper the subject will be considered from the various points of view which have occupied investigators, and which have been outlined above.

Methods.[2] The methods at present in use for determining the antitryptic value of serum are the fruit of a long process of evolution, the details of which need not here be sketched. There are essentially two methods in common use. The first of these, which was em-

[1] Referat to the American Association for Cancer Research, read at a meeting held in New York City, November 27, 1909.

[2] A more detailed critique of these methods may be found in a paper by the author, Archives of Internal Medicine, 1910, p. 109.

ployed by Brieger and Trebing,[3] makes use of plates of coagulated serum as the medium of digestion; on such plates a drop of solution of trypsin within twenty-four hours makes a visible depression or dell. A series of mixtures of the serum under examination, and of a standard solution of trypsin, is prepared, in which a constant quantity of the serum is added to increasing amounts of the ferment. Of each of these mixtures a loopful is transferred to the surface of the plates, which are then incubated at 55° for twenty-four hours. In this manner it is possible to determine in each case just how much of the trypsin solution can be totally inhibited by the standard quantity of serum, and so, to determine the "antitryptic titer" of the serum. The other method, known both as that of Fuld and of Gross,[4] employs a solution of casein as the medium of digestion. The serum in constant, and the trypsin solution in ascending, quantities, are added to a series of test-tubes containing equal amounts of casein in solution. At the end of two hours of incubation, the undigested casein is precipitated in all of the tubes by acidification; note is made of the lowest amount of trypsin which produces complete digestion, and this is taken to indicate the limit of the inhibitive activity of the serum. The results in both methods are expressed in figures, which denote the number of tenths of a cubic centimeter of trypsin inhibited. Through a preliminary determination of the inhibitory limits of normal serum it becomes possible to determine that certain serums have greatly diminished or greatly increased inhibitory power.

As regards the purely technical details of these methods, it may be said that both of them suffer from rather serious defects. The serum-plate method is open to the same objection which has been made to Mette's method of measuring the peptic activity of the gastric juice by means of the quantitative measurement of the amount of egg albumen which it is capable of digesting. Both egg albumens and the coagulated serums of different animals differ very considerably among themselves in digestibility, so that there is not the required constant basis of comparison (Klineberger and Scholz[5]). Furthermore, the visual appreciation of a minute depression on the surface of a serum plate is a very difficult and inexact procedure. When to these sources of error is added the fact that incubation is necessarily carried out at 55°—certainly far from the optimum for trypsin—and that bacterial contamination is a possible, though not a frequent, element of confusion, it may be understood that the method yields results only approximately accurate. The casein method is certainly far simpler and easier to manipulate. In addition to this, it has the added advantages of greater accuracy in the mixture of reagents, a normal temperature of incubation, and a period of experiment so short as to exclude bac-

[3] Berl. klin. Woch., 1908, p. 1041.

[4] Archiv f. exp. Path., 1907, p. 137.

[5] Deut. Archiv f. klin. Med., 1908, p. 319.

terial contamination; furthermore, the "end-points" of the readings are fairly sharp and accurate. Brieger[6] has objected, with regard to this method, that the classification of serums is more or less inexact; that the results are inconstant; and that the acid may produce a soluble acid albumin, and so obscure the "end-point" of the series of readings. None of these objections, however, is valid in fact; they are all purely theoretical. Numerous other objections have been made to the methods, the most important being that recently advanced by Marcus,[7] who was one of its originators. He states, as is well known to be the fact, that only a portion of the trypsin goes into solution, and hence maintains that there must be a considerable degree of variation in the strength of solutions presumed to be equal. He proposes to obviate this very serious difficulty by means of the use of glycerin extracts of the trypsin, which preserve their strength unaltered for considerable periods of time, and hence may be kept as standard stock solutions. Although apparently valid, this objection does not withstand the test of experiment. As shown by Dr. Feldstein and myself, the strength of a series of solutions of trypsin, made up independently with equal amounts of the ferment and salt solution, is astonishingly constant, and there is no need of the modification suggested by Marcus. The insoluble material is apparently evenly distributed as an inert impurity in the commercial trypsins.

The most serious objection which can be made to the method concerns the notation of results. This is apparently very simple. The results are stated in units, which represent the amounts of trypsin inhibited, in tenths of a cubic centimeter of the standard solution used. This mode of representing the results depends on the assumption that the amount of antitrypsin contained by serums is directly proportional to the quantity of trypsin which they are capable of inhibiting. In other words, the method presupposes that a serum of the titer 0.6 is twice as strong as one of 0.3. Neither the method of Brieger nor of Bergmann permits of an experimental tests of this hypothesis, but determinations made with the viscosimeter demonstrate that these relationships are distinctly not so simple as demanded by the theory. If an arithmetical series of solutions of trypsin be prepared, and the necessary inhibitory amount of serum determined for each member of the series, it is found that the quantitative intervals in the higher determination become increasingly larger. Consequently, the proportion indicated by the figures obtained by the serum or casein methods is entirely incorrect. Nevertheless, the relative antitryptic strength of the serums is correct, at least from a quantitative standpoint, and it is fair to accept the grouping of serums obtained by these methods as approximately accurate.

[6] Berl. klin. Woch., 1908, 1415, in report of discussion.
[7] Ibid., 1909, p. 156.

The viscosity method[8] previously referred to depends on the fact that the amount of digestion produced by trypsin in gelatin may be measured by determining the alteration in the viscosity of the latter medium. The effect of serum in controlling the activity of the trypsin is, of coure, very simply determined. The method offers certain very distinct advantages over those previously employed. Whereas, by the serum or casein methods, it is possible to make only one determination, namely, either the point of complete inhibition or of total digestion, the viscosimeter determines the degree of inhibition at any moment of time. The method, therefore, substitutes the use of a single mixture for that of a series, which is a very considerable gain in simplicity of technique. Moreover, the method is incomparably more flexible than those previously used, and permits of the determination of a large number of factors otherwise inaccessible to investigation.

The results obtained by the use of the two earlier methods have been strikingly concordant. Indeed, in comparing the findings yielded in a series of cases by both the serum and the casein methods, I found that the data were practically interchangeable. Brieger originally asserted that about 95 per cent. of the cases of cancer evinced a marked increase in the antitryptic value of their serum.[9] He subsequently found that in a large number of other conditions, including both acute infections and chronic wasting diseases, the same phenomenon could be observed, and he consequently concluded that all diseases associated with intense destruction of body protein produced this characteristic alteration in the plasma of the blood. Hence, although he continued to affirm the diagnostic value of the method in all cases of undetermined newgrowth, he reached the conclusion that in general all conditions of "cachexia"—using that term in the broad metabolic sense of wasting disease, either acute or chronic—were competent to produce it. Further investigation has, in general, given ample confirmation to these conclusions, if the term cachexia is interpreted in the peculiar sense in which it was used by Brieger. All observers are agreed that the great majority of cases of cancer give evidence of increased antitryptic value in the serum. In some series the percentage of positive results in cases of cancer ranges as high as 95 per cent.; in others as low as 70 per cent. It is, however, very frequently found in the acute infections, such as pneumonia, typhoid fever, sepsis, and polyarticular rheumatism; in chronic infections, notably tuberculosis; in diabetes and severe anemias; and in Graves' disease almost constantly. These data amply demonstrate that the change in the serum is not to be regarded as a characteristic biological response to the presence of newgrowths. They indicate that it is an evidence of pathological derangement of much wider

[8] Since the presentation of this paper, a preliminary report on the viscosity method has appeared, Feldstein and Weil, Proc. Soc. Exp. Biol. and Medicine, February, 1910.

[9] Berl. klin. Woch., 1908, pp. 1349 and 2260.

distribution. Furthermore, it seems misleading to consider the reaction as characteristic of conditions of cachexia, in view of the fact that this term must be extended so as to include a large number of conditions which can by no possibility be classified as cachectic. The general condition of nutrition of patients whose serum yields the reaction is frequently excellent, and could never be understood as cachectic. The theory of causation involved in the term "Kachexie Reaktion" will be subsequently discussed, but the term itself should certainly be allowed to fall into disuse

Not only under pathological conditions, however, does this reaction occur. It has been found to accompany and characterize certain processes which may be called physiological, although they denote a certain alteration in the normal course of metabolism. Its occurrence in the blood of infants has been investigated, and it has been found that such infants as are being nourished at the breast never display an increased antitryptic content of the blood (Reuss[10]). On the other hand, with the inauguration of artificial feeding, the reaction at once becomes prominent. In pregnancy it has been found that no antitryptic reaction occurs, but with the onset of labor it makes its appearance, and persists through the puerperium (Becker[11]).

It is evident that these findings may be discussed either from the standpoint of their diagnostic value, or as a biological phenomenon of purely theoretical interest. Diagnostically, the opinion of the various authors who have worked upon this problem is strikingly in accord. As a general diagnostic method, the increase in the antitryptic index occurs in too many conditions to have the value of a specific symptom. On the other hand, the absence of the antitryptic reaction in the blood may be taken generally as arguing against the existence of cancer. In the presence of a neoplasm of doubtful character, a positive reaction, in the absence of complicating conditions, notably tuberculosis, argues with a strong degree of probability in favor of the diagnosis of malignancy (Roche,[12] Hort,[13] Braunstein,[14] Bayly[15]).

The method has apparently stood the test of clinical experience, and has proved to be of distinct value when applied rigidly within the prescribed limits. As regards other conditions, Meyer[16] asserts that the reaction occurs with such regularity in cases of Graves' disease, that it may be relied upon in the diagnosis of the numerous obscure and abortive forms of the disease known as *formes frustes*. It has been claimed by Wiens[17] that the strength of the reaction has

[10] Wiener klin. Woch., 1909, p. 1171.
[11] Münch. med. Woch., 1909, p. 1363.
[12] Archives of Internal Medicine, 1909, p. 249.
[13] Brit. Med. Jour., 1909, p. 966.
[14] Deut. med. Woch., 1909, p. 573.
[15] Brit. Med. Jour., 1909, p. 1220. (Bayly measured digestion by electroconductivity.)
[16] Berl. klin. Woch., 1909, p. 1064.
[17] Deut. Archiv f. klin. Med., 1909, p. 62.

marked prognostic value in the acute infections, but this view is certainly erroneous.

Looked at as a biological phenomenon, the reaction suggests many problems, and has given rise to a considerable amount of research. The chemical nature of the antitrypsin, its character as a specific "immune body," its relationship to artificially produced antitrypsin, and the causes of its production, are subjects which demand elucidation, if the physiological significance and the pathological import of the reaction are to find an explanation.

The chemical basis of the antitryptic reaction has not been satisfactorily determined. It was originally asserted by Glaessner[18] that the antitrypsin was associated with the euglobulin fraction of the serum, which is that part of the serum globulins least soluble in water, and roughly corresponds to the fraction which comes down in dialyzing or on adding acetic acid to the diluted serum (Hedin[19]). Cathcart,[20] on the other hand, asserts that the globulins do not possess antitryptic action, but that this is characteristic of the albumin fraction, that is, the fraction precipitated between half and full saturation with ammonium sulphate. Schwarz[21] has reached the conclusion that the antitryptic fraction of the serum exists in the form of a lipoid. He found that he could inactivate antitryptic serums by washing out the lipoids with ether. Such serums could be reactivated by the addition of lecithin. Lecithin in salt solution emulsion, if added to serum, exercised considerable tryptic inhibition; if the mixtures were kept at 65° for one hour, this inhibitory activity was markedly enhanced, indicating that the inhibitory substance is a lipoid-albumin compound. Furthermore, he found by analysis that increase in the antitryptic titer of a serum was constantly associated with an increase in the amount of ether-soluble substances which it contained. These conclusions substantiate the earlier findings of Pribram. Interesting as are these data, they fail to support the contention that antitrypsin is a lipoid substance. It is well known, for example, that lipoids are essential to the activation of cobra venom in the production of hemolysis; nevertheless, it would be erroneous to consider the lipoid substance as the active hemolysin. Lipoids have been shown (Bang[22]) to play an analogous auxiliary role in many processes of immunity, while the essential factor, the active agent, is a protein. It is conservative to maintain this position with reference to antitrypsin, admitting meanwhile the possible importance of lipoids as subsidiary factors.

Is the so-called antitryptic action of the serum dependent on the presence of an "immune body," or is it an accidental property of the serum? This question is really of fundamental importance, though not at all easy to answer. It was found by Vernon[23] that

[18] Hofmeister's Beiträge, 1903, iv, 79.
[19] Jour. Physiol., 1903, p. 193.
[20] Ibid., 1904, xxxi, 496.
[21] Wien. klin. Woch., 1909, p. 1151.
[22] Ergebnisse d. Physiologie, 1909, p. 463.
[23] Jour. Physiol., 1904, p. 346.

egg albumin in solution inhibits the digestive action of trypsin very actively. But it is a still more striking fact that charcoal has been shown to act as an antitryptic agent, in a manner very similar to serum. The amount of inhibition is proportional to the quantity of charcoal, to the time of interaction, and to the temperature, just as it is in the case of serum. In fact, "the action of charcoal was found to agree with that of the tryptic antibody in all respects tried, and therefore the neutralizing effect, in all probability, is brought about in the same way in both cases" (Hedin[24]). It is perfectly apparent that neither egg albumen nor charcoal can in reality contain a true antitrypsin, and that, therefore, the effect observed is simply an accidental phenomenon. It is true of all human serums that they very notably inhibit the hemolytic effect of saponin; it would, however, be entirely unjustifiable to argue from this fact to the existence of an "antisaponin." These theoretical objections to the assumption of an "antitrypsin" in the serum have, unfortunately, not received recognition in the recent literature. On the other hand, it must be admitted that certain observations argue strongly in favor of a true antitrypsin, as against a general property of serum albumin, in the interpretation of tryptic inhibition. Chief among these is the alleged specificity of the antitryptic action. Specificity is, as is well understood, one of the most striking characteristics of all forms of antibody, and its absence may well be interpreted as a powerful argument in the negative. Eisner,[25] as the result of a series of tests made with the same serums against rennet, pepsin, emulsin, and cobra lipase, arrived at the conclusion that serum does not exhibit the properties of a general ferment inhibitor, but that it possesses a special and characteristic affinity for trypsin. This observation seems to indicate the existence of a specific antibody, a true antitrypsin. Glaessner has also, on insufficient evidence (Cathcart), asserted that the antitrypsin of serum is most active against the trypsin of the same species, and is somewhat specific even for various animal trypsins. The facts do not, however, appear to bear out these contentions. It has been possible in our laboratory, by means of the viscosimeter, to demonstrate that all human serums inhibit papain, which is a vegetable proteolytic ferment, in a constant ratio to the degree with which they inhibit trypsin. It seems, therefore, impossible to accept the specificity of the antitrypsin of the serum. The antitryptic function is exercised by an albuminous substance, thermolabile, indeed, like the true antibodies, but differing essentially from these in the lack of specificity. In view of this fact, and of certain other differences, the argument (Meyer[26]) in favor of a true antibody as the basis of this function of the serum loses very materially in credibility.

[24] Biochemical Journal, 1906, p. 484.
[25] Ztschr. f. Immunitätsforschung, 1909, ii, 650.
[26] Berl. klin. Woch., 1909, p. 2139.

This conclusion makes it very much simpler to dispose of the much debated problem as to the identity of the normal antitrypsin of the serum with that produced artificially in animals by the injection of trypsin. All antiferments hitherto produced by this method have been found to be characteristically specific. Thus, by the injection of rennet, Morgenroth[27] succeeded in producing an antirennin which powerfully inhibited the action of the injected ferment, but had no influence on vegetable rennet. The entire subject of antiferments is, however, in such a condition of confusion that it is almost impossible to draw any very definite conclusions. Achalme[28] produced an active antitrypsin by the injection of trypsin, but Landsteiner failed to reproduce this result. In our own laboratory, the injection of trypsin into guinea-pigs has been uniformly without effect. The most striking experiments are those recently reported from the Pasteur Institute on the result of the injection of pepsin and of papain. It has been shown by Cantacuzène and Jonescu[29] that when rabbits are immunized to pepsin by the injection of increasing doses, the serum responds by the production of an antibody capable of fixing complement, but possesses no increased antiferment action. Similarly, Pozerski[30] has shown that the serum of animals immunized to papain contains a specific precipitin, and an antibody which fixes complement in a characteristic fashion; but this very immunized serum is just as easily digested by the ferment as is normal serum. The natural antitrypsin, so-called, differs, therefore, in many important particulars from the antibody artificially produced by the injection of ferments into animals, and this fact constitutes an additional argument for regarding it as something essentially different from a true antibody.

The conception of antitrypsin as an antibody has, however, dominated practically all the theories which have hitherto been advanced in the attempt to explain it. In spite of the fact that it cannot properly be so regarded, these theories do not necessarily forfeit their validity. It is perfectly reasonable to assume that the serum may respond to a given stimulus by means of a protective mechanism which does not answer to the criteria characteristic of antibodies and amboceptors. It has generally, and very naturally, been assumed that the presence of antitrypsin in the serum is evidence of an effort on the part of the organism to protect itself against self-digestion. If this be the case, then a tryptic ferment should, theoretically, be present in the serum, and this has actually been demonstrated to be the fact by Hedin and by Delezenne;[31] it is, therefore, an important matter to determine its source of supply.

[27] Centralbl. f. Bakteriologie, 1899, p. 349.
[28] Annales de l'Institut Pasteur, 1901, xv, 736.
[29] Compt.-rend., de la Soc. de Biol. 1909, p. 53.
[30] Annales de l'Institut Pasteur, 1909, p. 205.
[31] Compt.-rend. de la Soc. de Biol., 1903, lv, 132.

There are four of such sources theoretically conceivable at present, namely, the pancreas, the leukocytes, the organs, and the new-growths. Each of these has had, and has, its champions, and each requires consideration. The pancreas, as a source of supply of trypsin, is a very obvious suggestion. Ambard[32] suggests that the antitryptic reaction is so marked in cases of gastric carcinoma, because this condition is associated with compensatory overactivity of the pancreas. There is, however, no evidence that the pancreatic trypsin is absorbed from the intestine, and circulates in the serum. Moreover, the explanation fails to explain the increase in Graves' disease, or the acute infections. The polynuclear leukocytes, as is well known, contain an active proteolytic ferment in most respects identical with trypsin. It has been urged that the constant disintegration of leukocytes must necessarily free a considerable amount of this ferment in the serum, and Hedin is of the opinion that the tryptic ferment which he succeeded in isolating from the serum actually represents the remnant of the intraleukocytic ferment. The view that the antitryptic reaction of serum is the manifestation of a response to the excessive disintegration of leukocytes has been urged and defended by Jochmann,[33] Wiens,[34] Weins and Schlecht,[35] Bittorf,[36] Landois,[37] and many others. As a result of the study of a large number of pathological conditions in which the differential leukocyte curve has been carefully plotted, and the antitryptic strength of the serum also has been repeatedly determined, it appears that there are certain definite relationships between these two factors. It has been quite satisfactorily demonstrated that with the onset of an infection the antitryptic index of the serum falls, and that with the progress of the infection it gradually rises again to the level of the normal, and then passes well beyond this to a highly increased index (Landois). Wiens, and Wiens and Schlecht, have shown that these fluctuations in the antitryptic index are accurately foreshadowed by variations in the leukocyte count, but only in so far as the polynuclear leukocytes determine these variations. The mononuclear cells do not play any role in influencing the index, and it is, therefore, of importance in the understanding of the interrelationship of these phenomena that the differential count should invariably be made. The explanation of these relationships is based on the well-known fact of the trypsin content of the polynuclear cells. With the onset of acute infections there is an immediate and rapid increase in the number of circulating polynuclear leukocytes. The inevitable destruction of a certain

[32] Sem. méd., 1908, p. 532.

[33] Münch. med. Woch., 1908, lv, 728; Hofmeister's Beiträge, 1908, p. 449.

[34] Deut. Arch. f. klin. Med., 1907, p. 456; Münch. med. Woch., 1907, p. 2637; Centralbl. f. Inn. Med., 1908, p. 773.

[35] Deut. Archiv f. klin. Med., 1909, p. 44.

[36] Ibid., 1907, xci, p. 212.

[37] Berl. klin. Woch., 1909, p. 440.

proportion of these cells frees an excessive amount of trypsin, which at once neutralizes all the available antitrypsin in the serum. Consequently, the antitryptic index falls well below normal, and may even disappear. This is the so-called negative phase of the antitryptic curve. The excess of trypsin in the blood, however, stimulates the production, or the mobilization, of fresh quantities of antitrypsin, which, in accordance with Ehrlich's interpretation of Weigert's laws of regeneration, are well in excess of the amount of trypsin to be neutralized. Consequently, there is a rapid rise in the antitryptic index. This is the so-called positive phase of the curve. A regulating mechanism of some kind tends, however, to keep the amount of antitrypsin in circulation only a little in excess of the trypsin, and this gives the value of the normal index. Fluctuations of this character, in which the leukocyte count and the curve of the antitryptic index pursue a parallel course, constitute a very striking feature of all infectious conditions. Wiens went so far as to assert that in such conditions a constantly increased antitryptic index was an omen of ill import, and augured the paralysis of the mechanism of defence, specifically the polynuclear leukocytes. Thaller[38] reached similar conclusions with reference to puerperal sepsis. In this belief they were unquestionably in error, inasmuch as the index is dependent quite as much on the amount of antitrypsin liberated by the body as on the leukocytes which represent the reaction to the disease. Indeed, the majority of acute infections, whether the body is in the ascendant or not, are associated with an increase in the antitryptic index. In addition to these clinical observations, there is ample experimental evidence (Miller[39]) that the injection of leukocytes, or of leukocyte extracts, into animals is followed, after a preliminary fall, by a marked rise in the antitryptic index. It may be seen from the preceding analysis that cases of myelogenous leukemia would not necessarily be associated with any notable variation in the antitryptic index, inasmuch as the regulatory mechanism maintains the index at its constant normal level. Jochmann, however, asserts that sudden myelocyte crises may so flood the blood with trypsin that the serum actually assumes digestive power, in place of its normal inhibitory function.

The preceding theory, interesting as it is, does not fully explain the phenomena. There are many conditions, such as diabetes, Graves' disease, and so forth, in which there is no increased production of leukocytes, yet the antitryptic index is constantly increased. If the leukocyte curve be admitted to afford a satisfactory explanation of the index in infectious conditions, there still remains a considerable number of conditions in which some other explanation must be discovered. In addition to the pancreas and the polynuclear leukocytes, there is another possible source of trypsin in the

[38] Berl. klin. Woch., 1909, p. 850.

[39] Zntralbl. f. Chir., 1909, p. 75.

body, namely, the cells of the tissues. It has now been abundantly shown that many, if not all, of the tissues contain proteolytic ferments, which are competent to break up these tissues outside of the body into a much simpler group of compounds. This process is known as autolysis, and the ferments in question are called autolytic ferments. Although they are actively proteolytic, they appear to differ in certain particulars from true trypsin. Thus, the end-products indicate that an ereptic ferment is almost certainly at work (Vernon[40]). Furthermore, it has been asserted by Jacoby[41] that these ferments are adapted specifically to the proteolysis only of the organs in which they occur, a characteristic which, *if well founded* (Beebe),[42] would sharply differentiate them from true trypsin. In spite of these objections, there are certain facts which indicate their possible relationship with the antitryptic phenomenon of the serum. In the first place, it is known that the injection of tissue other than the pancreas may induce a rise in the antitryptic index. Further, serum exercises an anti-autolytic (Baer and Loeb[43]), just as it does an antitryptic, power. Finally, it has been shown by Shaffer and Buxton,[44] and others, that glycerin extracts of the various organs, including the muscles, are capable of displaying marked proteolytic powers, when tested, for example, upon milk agar plates. The difficulty in all observations of this kind consists in excluding the leukocytes themselves. If it must be admitted that the conditions of experimentation have not yet permitted a final decision as to the character of the ferments contained in the organs, the fact still remains that the tissues do contain a proteolytic ferment, probably very similar to trypsin. In view of this fact, the theory has been advanced that the destruction of body protein from any cause would tend to free the proteolytic ferments contained in the cells, and that the somatic reaction would liberate an excess of antitrypsin in the serum. This theory, it will be seen, is simply an expansion of the leukocyte theory, inasmuch as the leukocytes may be considered as a type of cell distinguished by their increased content of proteolytic ferments. There can, indeed, be no question that it offers an explanation for the increased index in a large number of diseases for which the leukocyte theory is entirely inadequate, such as the marasmus of infants (Lust[45]). This includes not only conditions such as Graves' disease, diabetes, and chronic tuberculosis, but certain acute infections, such as typhoid fever, not associated with leukocytosis. There is, however, one very weak point in this theory, in spite of the fact that it apparently harmonizes with clinical conditions, and this is the unwarranted assumption that heightened protein metabolism is necessarily associated with the liberation

[40] Intracellular Enzymes, 1909.
[41] Ztschr. f. Physiol. Chem., 1901, vol. xxxiii.
[42] Boston Med. and Surg. Jour., 1907.
[43] Arch. f. exp. Path., 1905, p. 1; 1906, p. 68.
[44] Jour. Med. Research, 1905.
[45] Deut. med. Woch., 1909, p. 1901.

of intracellular ferments. The advocates of the theory have devoted a great deal of effort to the support of this assumption. It has been asserted (Fürst) that starvation, with its accompanying cellular destruction, raises the index, but this again has been denied (Meyer). The effect of cellular poisons, such as pilocarpin, phosphorus, and potassium cyanide, has been tested, but without the expected result in raising the index. The kidneys have been tied off, and allowed to necrose *in situ*, but in spite of the presumptive absorption of the cellular constituents including ferments, no rise in the antitryptic index was observed. It must be admitted, therefore, that experimental data fail to give any support to this theory.

The increase of the index in cancer is attributed to the same cause, namely, the liberation of the intracellular ferments, which are well known to be very active in cancerous growths (Bamberg[46]). With the frequent tendency to necrosis in tumors, even if only in microscopic areas, there would seem to be abundant opportunity for the absorption of ferments. But here, again, the absence of evidence that such a process actually does occur is a fundamental flaw in the theory.

To sum up, the origin of the hypothetical trypsin which is supposed to act as a stimulant for the production of antitrypsin, or, technically speaking, as antigen, is as yet undetermined. It may, conceivably, arise in the pancreas, or in the leukocytes, or in the tissue cells, or in the newgrowths, or in each one of these, under varying circumstances, but actual evidence that it does so arise is at the present time an absolute necessity for the establishment of the theory. The very first essential is to determine whether or not the trypsin, or proteolytic ferment of the blood is increased, in accordance with the assumption of the hypothesis, in the conditions which give rise to an increase in the antitryptic index. At the present time, no method seems to be available for this purpose.

THE WASSERMANN AND NOGUCHI COMPLEMENT-FIXATION TEST IN LEPROSY.[1]

By Howard Fox, M.D.,
of new york.

The first to obtain a positive Wassermann reaction in a case of leprosy was Eitner[2] in 1906. A similar report was made by Weichsel-

[46] Berl. klin. Woch., 1908, pp. 1396 and 1673.

[1] Read at a meeting of the Medical Society, of the State of New York, January 24, 1910.

[2] Ueber den Nachweis von Antikörpern im Serum eines Leprakranken mittels Komplementablenkung, Wien, klin. Woch., 1906, No. 15, p. 1555.

mann and Meier[3] nearly two years later. Since then it has been found by a number of observers that leprosy quite frequently gives a positive reaction. In testing 26 advanced cases of the disease, Slatineanu and Danielopolu[4] found 20 strongly positive, 4 moderately positive, and 2 weakly positive reactions. Jundell, Almkvist, and Sandman,[5] in a series of 26 cases obtained 4 strong and 4 moderately positive reactions. In 2 cases the result was unsatisfactory, while in the remaining 16 cases the reaction was negative. Of the positive cases, 5 were of the tubercular and 3 of the maculo-anesthetic type. From this Sandman concludes that the occurrence of the reaction does not depend upon the type of the disease, whether tubercular or anesthetic. Meier[6] on the other hand in a series of 28 cases, found positive reactions only in the tubercular type of leprosy. All of the anesthetic cases gave negative reactions. The number of cases of each type was unfortunately not stated. Similar results were obtained by Bruck and Gessner[7] who found positive reactions in 5 out of 7 tubercular cases and negative reactions in 3 anesthetic cases. Positive reactions have also been obtained by Gaucher and Abrami[8] in 8 cases and by Frugoni and Pisani[9] in 9 out of 11 cases of leprosy, the type of the disease, however, not being stated.

Eitner[10] was also the first to obtain complement-fixation in leprosy, using an extract of leprous tissue as antigen. Similar results were later reported by Slatineanu and Danielopolu,[11] Gaucher and Abrami Sugai,[12] Pasini,[13] and by Frugoni and Pisani. It was also found by Slatineanu and Danielopolu[14] that complement could be fixed by leprous serum employing tuberculin as antigen. Complement-fixation in leprosy was also obtained by Frugoni and Pisani by using tuberculin, tubercle bacilli, and extracts of sarcoma and carcinoma as antigen.

It has been my privilege during the past six months to have em-

[3] Wassermannsche Reaktion in einem Falle von Lepra, Deut. med. Woch., 1908, No. 31, p. 1340.

[4] Réaction de fixation avec le sérum et le liquide céphalo-rachidien des malades atteints de lèpre en présence de l'antigène syphilitique. Séances et mém. d. l. Soc. d. biol., 1908, xi, p. 347.

[5] Wassermann's Syphilisreakton bei Lepra, Zntralbl. f. innere Med., 1908, No. 48, p. 1181.

[6] Zur Technik und klinischen Bedeutung der Wassermannschen Reakton., Wien. klin. Woch., 1908, No. 51, p. 1765.

[7] Ueber Serumuntersuchungen bei Lepra, Berl, klin. Woch., 1909, No. 13, p. 589.

[8] Le séro-diagnostic des formes atypiques de la lepre, 1909, viii, p. 152.

[9] Vielfache Bindingseigenenschaften des Komplements einiger Sera (Leprakranken) und Ihre Bedeutung. Berl. klin. Woch., 1909, No. 33, p. 1530.

[10] Zur Frage der Anwendung der Komplementbindungsreakton auf Lepra, Wien. klin. Woch., 1908, No. 20, p. 729.

[11] Sur la présence d'anticorps spécifiques dans le sérum des malades atteinte de lèpre, Séances et mém d. l. Soc. de biol., 1908, xi, p. 309.

[12] Zur klinisch-diagnostischen Verwertung der Komplementbindungs methode bei Lepra. Archiv. f. Dermatol. u. Syph., 1909, p. 313.

[13] Sulla reazione della deviazione del complemento nella lepra. Reviewed in Giorn. Ital. d. Malatt. Vener. e.d. pelle, 1909, No. 111.

[14] Réaction de fixation dans la lèpre en employant la tuberculine comme antigene, Seances et mem. d. l. Sec. de Biol., 1908, lxv, p. 530.

ployed the Wassermann reaction in 60 cases of leprosy. Fifteen of these cases were seen in various clinics and hospitals in New York City. The remaining forty-five were seen during a recent visit to the Leper Home in Louisiana, an institution under the direction of Dr. Isadore Dyer of New Orleans. All of these 15 cases with one exception were tested by both the regular Wassermann and the Noguchi methods, the results in all cases being identical. The cases in Louisiana were tested alone by the more convenient method of Noguchi, owing to lack of time at my disposal. The technique used was the same as that described in some of my previous communications[15] and will be here omitted for the sake of brevity. It may, however be remarked that the antigen used in the Wassermann test was an alcoholic extract of syphilitic liver. The antigen used in the Noguchi[16] test consisted of acetone insoluble lipoids. The patient's serum in the Noguchi method was used in active condition. All of the cases examined were undoubted lepers, many of them having been under observation for years. No history of syphilis was obtainable in any case. Certainly no lesions were seen in any patient that could have been regarded as syphilitic.

To summarize the results, of the 38 cases of the tubercular and mixed type, the reaction was negative in 7, weakly positive in 3, positive in 21, and strongly positive in 7 cases. Of the 22 maculo-anesthetic and purely anesthetic cases, the reaction was negative in 19, strongly positive in 1, and positive in 2 cases.

It may be of interest to add that beside the 15 cases of leprosy examined in New York, I have also seen or personally known during the past six months, of 7 other cases (3 of Dr. J. McF. Winfield, and one each of Drs. Wm. B. Trimble, M. B. Parounagian, F. M. Dearborn, and G. H. Fox). It will doubtless seem surprising to some that there should have been so many cases of leprosy in New York City during such a short space of time.

Cases of Tubercular and Mixed Type with Positive Reaction.[17] Case I.—Patient of Dr. S. Dana Hubbard, service of Dr. Jackson, Vanderbilt Clinic. I. W., West Indian negress, aged thirty-three years. Advanced case of tubercular type. Duration of disease two years. Reaction: strongly positive.

Case II.—Patient of Dr. G. H. Fox, New York Skin and Cancer Hospital. S.V., man, aged forty years, born in Russia. Active fairly advanced case of mixed type. Duration, two years. Reaction: Strongly positive.

[15] The Principles and Technique of the Wassermann Reaction and its Modifications. Med. Record, 1909, p. 421; a Comparison of the Wassermann and Noguchi Complement Fixation Tests, Jour. Cutan. Dis., 1909, p. 338; The Wassermann Reaction (Noguchi Modification) in Pellagra, New York Med. Jour., 1909, p. 1206.

[16] On Non-specific Complement-fixation, Proceed. Soc. Exper. Biol. and Med., December, 1909.

[17] Cases not designated by the name of physician and name of clinic where treated, were all seen at the Louisana Leper Home in the service of Dr. Isadore Dyer.

Case III.—Patient of Dr. G. H. Fox, New York Skin and Cancer Hospital. P. N., man, aged forty-two years, Italian, Armenian. Advanced case of mixed type. Duration said to be two years. Reaction: Strongly positive.

Case IV.—Patient of Dr. G. H. Fox, New York Skin and Cancer Hospital. S. V., man, aged twenty-seven years, Italian. Very marked active case of tubercular type. Duration, three years. Reaction: Positive.

Case V.—Patient of Dr. L. Duncan Bulkley, New York Skin and Cancer Hospital. R. R., Russian woman, aged sixty years. Advanced case of mixed type. Duration, ten years. Reaction: Positive.

Case VI.—Patient of Dr. J. McF. Winfield, Kings County Hospital. C. W., negro, aged twenty-six years, born in United States. Mixed type of moderate severity, of eight years' duration. Reaction: Positive.

Case VII.—Patient of Dr. J. McF. Winfield, Kings County Hospital. L. M., man, aged about fifty years, Russian. Advanced case of mixed type. Duration, about twenty years. Reaction: Positive.

Case VIII.—Patient of Dr. F. M. Dearborn, Metropolitan Hospital. P. L., Chinaman, aged thirty-nine years. Advanced active case of mixed type. Duration six years. Reaction: Positive.

Case IX.—Patient of Dr. Wm. S. Gottheil, City Hospital. Chinaman, aged twenty-nine years. Moderate case of tubercular type, of four years' duration. Reaction: Weakly positive.

Case X.—Patient of Dr. Wm. S. Gottheil, City Hospital. E. G., man, aged twenty-seven years, born in Russia. Mild case of tubercular type. Duration three and a half years. Reaction: Positive.

Case XI.—Patient of Dr. L. Oulman, German Hospital. L. T., woman, aged twenty-four years, born in Russia. Case of mixed type of moderate severity. Duration, nine years. Reaction: Strongly positive.

Case XII.—Colored woman, aged fifty-seven years, active tubercular case. Duration of disease four years. Reaction: Positive.

Case XIII.—White woman, aged forty-eight years. Advanced case of mixed type. Duration fourteen years. Reaction: Positive.

Case XIV.—White woman, aged twenty-seven years. Case of mixed type. Duration, seven years. Patient improving. Reaction: Weakly positive.

Case XV.—White woman, aged fifty years. Mixed type of the disease in an advanced stage. Reaction: Positive.

Case XVI.—White woman, aged forty years. Advanced and active case of mixed type. Reaction: Positive.

Case XVII.—Colored woman, aged fifty years. Advanced case of tubercular type. Duration of disease, three years. Reaction: Positive.

Case XVIII.—White boy, aged sixteen years. Case of tubercular type. Duration, nine years. Reaction: Positive.

Case XIX. White man, aged forty-eight years. Incipient type, in which the disease is active. Duration, five years. Reaction: Strongly positive.

Case XX.—White man, aged forty-five years. Advanced case of mixed type. Duration, seventeen years. Reaction: Positive.

Case XXI.—Colored man, aged forty-eight years. Advanced case of mixed type, in which process is stationary. Duration, four years. Reaction: Positive.

Case XXII.—Colored man, aged thirty-seven years. Active case of tubercular type. Duration, five years. Reaction: Weakly positive.

Case XXIII.—Colored man, aged fifty years. Advanced case of mixed type. Disease active. Duration, five years. Reaction: Strongly positive.

Case XXIV.—White boy, aged eighteen years. Terminal case of tubercular type. With active lesions. Duration, twelve years. Reaction: Positive.

Case XXV.—White boy, aged nineteen years. Advanced case of mixed type. Duration, five years. Reaction: Positive.

Case XXVI.—White boy, aged sixteen years. Advanced case of mixed type. Duration, four years. Reaction: Positive.

Case XXVII.—White boy, aged twenty years. Incipient case of mixed type, relapsing after apparent cure. Duration, nine years. Reaction: Strongly positive.

Case XXVIII.—Colored man, aged forty-two years. Terminal stage of mixed type. Duration, three years. Reaction: Positive.

Case XXIX.—White woman, aged thirty-five years. Advanced active case of mixed type. Duration, fourteen years. Reaction: Positive.

Case XXX.—White woman, aged fifty-seven years. Advanced case of mixed type, tubercles having disappeared. Duration, twenty years. Reaction: Positive.

Case XXXI.—White man, aged forty years. Terminal stage of mixed type. Duration, eight years. Reaction: Positive.

Cases of Tubercular and Mixed Type with Negative Reaction. Case XXXII.—Patient of Dr. Wm. S. Gottheil, City Hospital. H. S., man, aged thirty-three years, born in the United States. Case of mixed type of moderate severity. Duration, ten years. Reaction: Negative.

Case XXXIII.—Patient of Dr. F. M. Dearborn, Metropolitan Hospital. J. M., man, aged fifty years, born in Russian Poland. Case of mixed type. Very few lesions at present, though formerly well marked. Duration of disease not known. Has been in leper ward for the past six years. Reaction: Negative.

Case XXXIV.—White man, aged twenty-eight years. Mixed type. Patient improving. Duration of disease, eighteen years. Reaction: Negative.

Case XXXV.—White man, aged twenty-one years. Incipient case of mixed type, which is improving. Duration, six years. Reaction: Negative.

Case XXXVI.—White man, aged twenty-four years. Advanced case of mixed type. Disease active. Duration, eighteen years. Reaction: Negative.

Case XXXVII.—Colored man, aged twenty-six years. Terminal case of mixed type. Duration, probably five years. Reaction Negative.

Case XXXVIII.—White woman, aged forty-three years. Case of mixed type, improving, tubercles having disappeared. Duration, twenty years. Reaction: Negative.

Cases of Maculo-anesthetic Type with Positive Reaction. Case XXXIX.—Patient of Dr. G. H. Fox, New York Skin and Cancer Hospital. T. D., girl, born in Key West, Florida. Maculo-anesthetic case of one year's duration. Reaction: Positive.

Case XL.—Colored woman, aged sixty-four years. Incipient anesthetic case. Duration three years. Reaction: Strongly positive.

Case XLI.—Colored woman, aged fifty-nine years. Muculo-anesthetic case, improving. Duration, two years. Reaction: Positive.

Cases of Maculo-anesthetic Type with Negative Reaction. Case XLII.—Patient of Dr. J. McF. Winfield, Kings County Hospital. J. D., West Indian negro, aged twenty-nine years. Maculo-anesthetic type. Duration, about twenty-three years. Reaction: Negative.

Case XLIII.—White girl, aged seventeen years. Incipient case of maculo-anesthetic type. Duration, fourteen years. Reaction: Negative.

Case XLIV.—White woman, aged about sixty years. Anesthetic type in advanced stage. Duration of the disease, unknown. Reaction: Negative.

Case XLV.—White woman about fifty years of age. Advanced case of anesthetic type. Duration of the disease, unknown. Reaction: Negative.

Case XLVI.—White woman, aged about fifty years. Incipient maculo-anesthetic case. Duration unknown. Reaction: Negative.

Case XLVII.—White woman, aged eighty-seven years. Incipient case of maculo-anesthetic type. Duration, five years. Reaction: Negative.

Case XLVIII.—Colored woman, aged fifty-three years. Advanced anesthetic case. Duration, twenty-seven years. Disease checked. Reaction: Negative.

Case XLIX.—Colored woman, aged about sixty years. Advanced anesthetic case, the disease being stationary. Duration, fifteen years. Reaction: Negative.

Case L.—White woman, aged thirty-four years. Maculo-anesthetic case. Former tubercles have disappeared. Duration, eight years. Reaction: Negative.

Case LI.—White boy, aged nineteen years. Advanced case of anesthetic type. Duration, nine years. Reaction: Negative.

Case LII.—White man, aged forty years. Maculo-anesthetic type, improving. Duration, fourteen years. Reaction: Negative.

Case LIII.—White girl, aged twelve years. Incipient case of maculo-anesthetic type. Duration, four years. Reaction: Negative.

Case LIV.—Colored boy, aged nine years. Incipient case of maculo-anesthetic type. Duration, four years. Reaction: Negative.

Case LV.—White man, aged forty-three years. Advanced case of anesthetic type. Duration, thirty years. Disease arrested. Reaction: Negative.

Case LVI.—White man, aged fifty-four years. Incipient case of anesthetic type. Duration ten years. Reaction: Negative.

Case LVII.—White man, aged fifty-eight years. Terminal case of anesthetic type. Duration, thirty years. Reaction: Negative.

Case LVIII.—White man, aged fifty-six years. Terminal stage of anesthetic type. Duration, thirty years. Reaction: Negative.

Case LIX.—Chinaman, aged seventy-five years. Anesthetic case of thirteen years' duration. Reaction: Negative.

Case LX.—White man, aged forty-six years. Advanced anesthetic case. Patient claims to have been discharged cured from a Norwegian hospital twenty years ago. Duration, twenty-five years. Reaction: Negative.

Conclusions. 1. A positive Wassermann reaction is frequently obtained in cases of leprosy giving no history or symptoms whatever of syphilis.

2. The reaction is at times very strong, inhibition of hemolysis being complete.

3. The reaction occurs chiefly in the tubercular and mixed forms of the disease, especially in advanced and active cases.

4. In the cases of the maculo-anesthetic and purely trophic type the reaction is generally negative.

5. The value of the test is not affected in the slightest by the results found in leprosy.

In closing, I desire to express my thanks to Dr. Isadore Dyer for kindly putting at my disposal the splendid material of the Louisiana Leper Home. I also wish to thank Dr. Ralph Hopkins, the attending physician to the Leper Home for aid in obtaining case histories. For the material in New York I am indebted to the physicians whose names have been mentioned in the text.

THE EFFECT OF TUBERCULOSIS ON INTRATHORACIC RELATIONS.[1]

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The following preliminary report deals with the changes brought about in intrathoracic relations as shown by skiagrams of cases of pulmonary tuberculosis, studied clinically before and after the taking of the *x*-ray plates. I will confine myself simply to the more salient features of this study, which deals with the alterations in the position of aorta, heart, and diaphragm. The presence or absence, of enlarged bronchial glands, and of calcareous infiltration in the costal cartilages of the ribs is also briefly considered.

When such a study is carried out with technical precision, it would seem to leave little room for error; because with the use of the modern tubes and coils, and instantaneous exposures, the resulting skiagrams are so clear cut and definitive as to give an admirable geographical chart. I believe that such a study gives more satisfactory results in regard to the position of the organs in life than those obtained by autopsy, for in the latter there is the postmortem change, due to alteration in intrathoracic pressure or to other strictly postmortem influences; there is also the trauma of the autopsy and consequent derangement of relations, and finally there is the question of elapsed time. I feel further that purely clinical studies of this nature, when unconfirmed by röntgenology, are without much accuracy or value, except in so far as in certain instances the personal equation of the investigator has given them such.

The value of röntgenography in the study of pulmonary tuberculosis, as supplementary to physical examination, is so generally recognized as to need no exposition. I shall not discuss the use of the *x*-rays from the standpoint of early diagnosis, but in their relations to the later pathological changes. Röntgenology must not, of course, be considered as having solved the problems of physical diagnosis of the chest, but it helps to elucidate them and to confirm the clinical findings. It has not and never can supplant nor minimize the importance of the time-honored clinical methods, but should on the contrary serve a useful purpose in stimulating more exact methods, because the possibilities of physical diagnosis are extended from the information and suggestions gleaned from the *x*-rays.

The cases from which this study is made were skiagraphed by

[1] Read at the XVI International Medical Congress, Budapest, August, 1909.

Dr. Charles Lester Leonard, of Philadelphia, and were in some instances patients from the Pennsylvania State Dispensary, No. 21, for Tuberculosis, and in others from private practice. The majority of the cases were moderately advanced and advanced cases (Class II and III of the National Association classification), though the only selection used was in the financial ability of the patient to bear the expense of the skiagrams. The number here reported is too small (that is, 60 cases) to warrant me in drawing any very definite conclusions, but the results are at least suggestive, and the conclusions which I do present relate only to this series.

I shall not attempt at this time to discuss the data gleaned from this study, in the light of present knowledge or views, but shall content myself with merely recording the details noted, pointing out where in certain instances the conclusions drawn are at variance with the views or opinions of others.

The Aorta and Heart. It should be noted that quite frequently in advanced pulmonary tuberculosis the aorta is displaced as well as the heart and in the same direction, usually to the right. In marked displacements of the heart this is the rule (Cases II, IV, VI, XII, XIII, XXV, XXVII, XXVIII, XLVI, XLVII, LII, LIX). Rarely the aorta may be drawn out of position, while the heart is unaffected (Case XXXIII). The error is sometimes made of interpreting the physical signs of a displaced aorta, as being those of enlarged glands or of aneurysmal dilatation. With an area of dulness to the right of the sternum in the second or third interspace and much displacement of the heart, the conclusion that the aorta is displaced is warranted, in the absence of definite signs of aneurysm. Rarely an aneurysmal dilatation of the aorta may be present.

It is noteworthy that in the present series, many of which were advanced or far advanced cases, the heart in the majority of instances was not displaced. There seems no doubt about this conclusion, and I therefore feel that those who hold that displacement of the heart is a reasonably constant sign or accompaniment of pulmonary tuberculosis are in error. Turban makes the statement that "it is exceptional to find the heart in its normal position in advanced chronic tuberculosis,"[2] while Pottenger says that displacement of the heart is a "typical and cardinal symptom" of tuberculosis of the right apex.[3]

In 63.33 per cent. of the cases the heart was not displaced (that is, in 38 cases, I, III, V, VII, VIII, X, XI, XIII, XIV, XVI, XVII, XIX, XX, XXI, XXII, XXIII, XXIV, XXVI, XXVIII, XXIX, XXX, XXXII, XXXIII, XXXV, XXXVII, XL, XLI, XLIV, XLVIII, XLIX, L, LI, LIII, LIV, LV, LVI, LVII, LX).

Absence of displacement is much more common, in fact almost

[2] Diagnosis and Treatment of Pulmonary Tuberculosis, 1908

[3] Diagnosis of Tuberculosis of the Lungs, 1906.

the rule, in acute infiltrations and consolidations before fibrosis and contraction have taken place. In many instances even when the lesions were widespread and often destructive, the heart was not displaced. (Cases I, III, VIII, XI, XIII, XIV, XVI, XVII, XX, XXII, XXIII, XXIV, XXXII, XXXIII, XXXV, XLVIII, XLIX, L, LI, LVII). In Case XIII, with cavitation at the right apex and infiltration of the entire upper lobe, the heart was not displaced, due to pericardial adhesions over apex which could be plainly seen. In Case XXIII, with large cavities in both apices and much fibrosis, the heart was not displaced, possibly on account

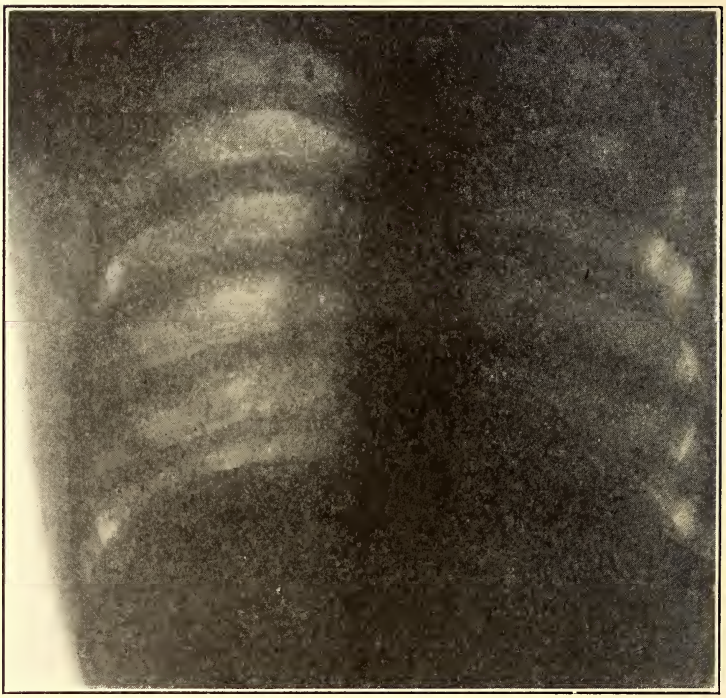

FIG. 1.—Case XXIV. Left-sided localized pneumothorax without displacement of the heart. The case belongs to Class III of the National Association classification.

of the symmetrical character of the lesions, or possibly from adhesions. In Case XXIV (Fig. 1), with left-sided localized pneumothorax, the heart was not displaced. In Case XXXIII, with a large cavity on the right and complete consolidation of the right upper lobe, and moderate infiltration on the left, the heart was not displaced. In the following far-advanced cases with cavity the heart was not displaced: Cases XI, XIII, XIV, XXIII, XXIV, XXXIII, XLVIII, XLIX, L.

The heart was displaced in 36.66 per cent. of this series, in 15 cases to the right (Cases II, IV, XII, XV, XXV, XXVII, XXXVI,

XXXVIII, XLIII, XLV, XLVI, XLVII, LII, LVIII, LIX); in 3 cases to the left (Cases VI, IX, XXXI); and in 4 cases in the anteroposterior position (Cases XVIII, XXXIV, XXXIX, XLII), to be described later. I cannot, therefore, agree with the statement of Lawrason Brown[4] that "marked displacement of the heart occurs much more frequently to the left than the right."

On the contrary, when the pulmonary lesions are of fairly symmetrical character on both sides, the heart is more commonly displaced to the right than to the left (Cases II, XXV, XXXVI, LII, LVIII,

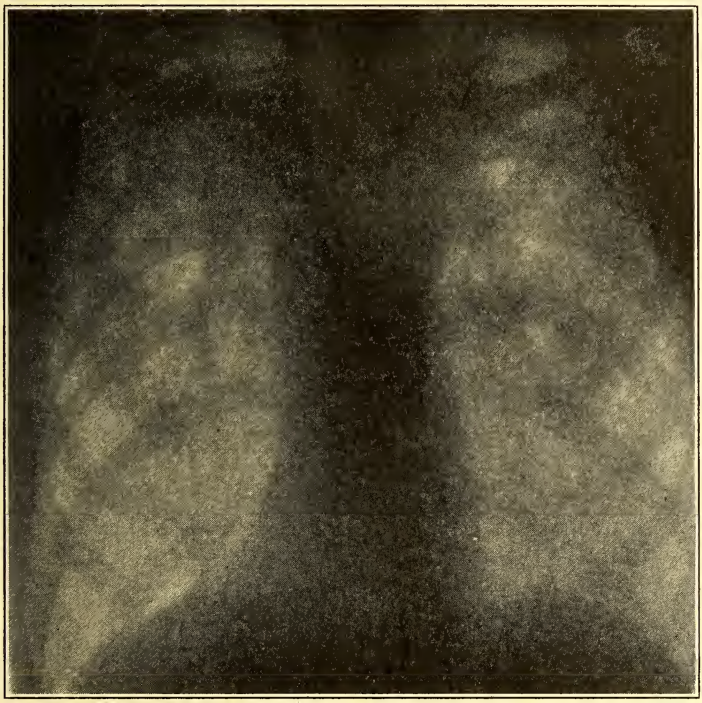

FIG. 2.—The heart in the anteroposterior position. Destructive lesions at both apices. The case belongs to Class III of the National Association classification.

LIX.) In these cases there is usually evidence to show that the primary and older lesion is on the right. When the lesion is more extensive on the left the heart is not so regularly displaced nor to the same extent, as in corresponding right-sided lesions (Cases I, III, XIV). In Case XIV, with cavity in the left apex and consolidation of the lung below, and moderate infiltration of the right apex, the heart was not displaced. Rarely the fibrosis of the lungs and pleuræ may be so great or of sufficient density to obliterate the boundaries of the heart (Case IX).

[4] AMER. JOUR. MED. SCI., 1908.

In 4 cases the heart occupied, what for want of a better term, I have called the anteroposterior position (Cases XVIII, XXXIV, XXXIX, XLII). In this position (Figs. 2 and 3) the heart assumes a long narrow appearance, as if it were turned upon its vertical axis. It should be noted that in all these cases, there were far advanced destructive lesions on both sides, and it might appear that the combined effect of the traction exerted under these conditions, had resulted in drawing the heart upward and inward, thus causing the apex to swing around. In long narrow chests the heart assumes a more

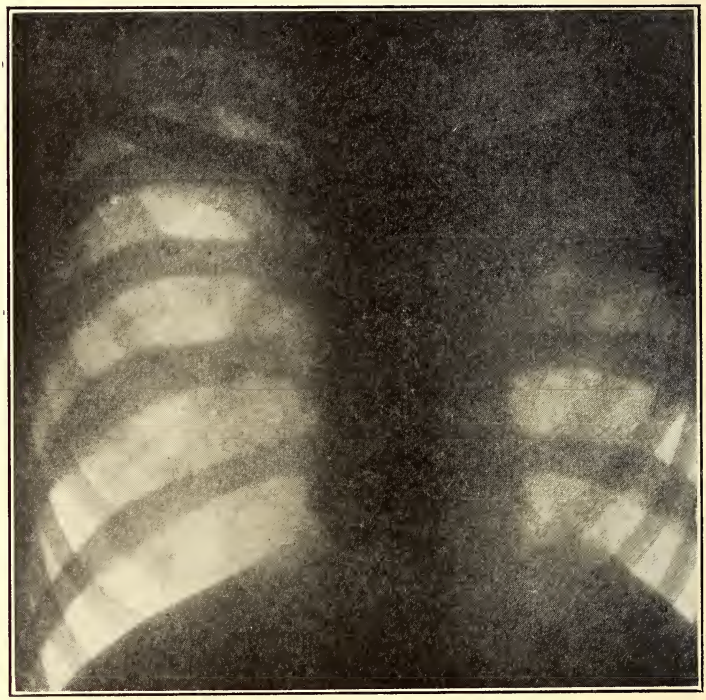

FIG. 3.—The heart in the anteroposterior position. Destructive lesions at both apices. The case belongs to Class III of the National Association classification.

oblique position. The angle formed with the liver on the right is less acute and the left boundary is appreciably more vertical. The heart also assumes an appreciably more oblique position during deep inspiration.

In many cases the skiagrams show an interesting feature which is not demonstrable clinically, namely, that during systole of the heart there is an area between the lower boundary of the heart and the diaphragm of about the extent of one centimeter, which distinctly transmits the *x*-rays. The limits or extent of the cardiac excursion may also be seen in many cases. In four cases, distinct

pericardial adhesions could be seen (Cases VII, XI, XIII, XVIII) The heart appeared normal in size both in the *x*-ray plates and to physical examination in all the cases with four exceptions (Cases XIX, XXIII, LVI, LIV). In three of these the enlargement was practically confined to the right side, and the lungs were markedly emphysematous. In Case XIX there was a general hypertrophy. There was no evidence of an organic valvular lesion in the series, though in some cases soft systolic murmurs were audible in the mitral and pulmonary areas.

The Diaphragm. The skiagrams were taken under full inspiration. In 29 cases (48.33 per cent.) the diaphragm was unaffected by the pulmonary lesion (Cases I, III, V, VII, X, XI, XIII, XVII, XIX, XX, XXI, XXIV, XXVI, XXVIII, XXIX, XXX, XXXII, XXXV, XL, XLI, XLIV, XLVIII, LI, LIV, LV, LVI, LVII, LX); the diaphragm was affected in this series in 51.66 per cent. of the cases. In 13 cases it was elevated on the right side (Cases II, VIII, XII, XV, XVI, XXII, XXV, XXXVIII, XLIII, XLV, XLIX, L, LVIII); in 5 cases on the left (Cases VI, XIV, XXVII, XXXI, LIII); in 7 cases it was elevated on both sides (Cases XVIII, XXIII, XXXIII, XXXIV, XXXVI, XXXIX, XLII); and in 6 cases it was not visible or determinable on account of the density of the adjacent involvement of the lungs and pleura (Cases IV, IX, XLVI, XLVII, LII, LIX).

In every case in which the heart was displaced the diaphragm was elevated on the side toward the displacement, and in the cases in which the heart assumed the anteroposterior position the diaphragm was elevated on both sides. There was one exception to this rule which does not properly apply as such, but in Case XXVII, which had been operated on for left-sided empyema some years previously, there was collapse of the chest wall, with consequent dragging upward of the diaphragm on that side, while the heart was displaced to the right.

The diaphragm was affected in 9 cases in which the position of the heart was normal (Cases VIII, XIV, XVI, XXII, XXIII, XXXIII, XLIX, L, LIII) (Fig. 4). In other words, the diaphragm was more sensitive to, or affected by, the presence of a pulmonary lesion than the heart in 15 per cent. of the cases. This was true in 5 advanced cases (Cases VIII, XIV, XXXIII, XLIX, L), as well as in 4 of the earlier cases (XVI, XXII, XXIII, LIII); and yet in 10 advanced cases in which one would have expected to find the diaphragm affected, it was not apparent (except in limitation of pulmonary excursion), either to physical examination or in the plates (Cases I, III, XI, XIII, XVII, XXIV, XXXV, XLVIII, LI, LVII). Thus in cases of relatively slight involvement the diaphragm may be elevated on the affected side (Case XXII, as type); while in cases with marked involvement and even cavitation the diaphragm may not be elevated (Case XIII as a type).

Thus the diaphragm had responded, in change of position, to the pulmonary lesion in only half the cases.

The Peribronchial Lymph Nodes. In every case in this series the cervical glands were enlarged to palpitation. It would seem probable that the peribronchial glands would also be affected in all cases, though this could not be deduced from the skiagrams. In 51.66 per cent. of the cases enlarged glands could be seen in the plates (Cases V, VII, VIII, X, XIII, XVII, XVIII, XIX, XX, XXI, XXII, XXIII, XXVI, XXVII, XXVIII, XXIX, XXX,

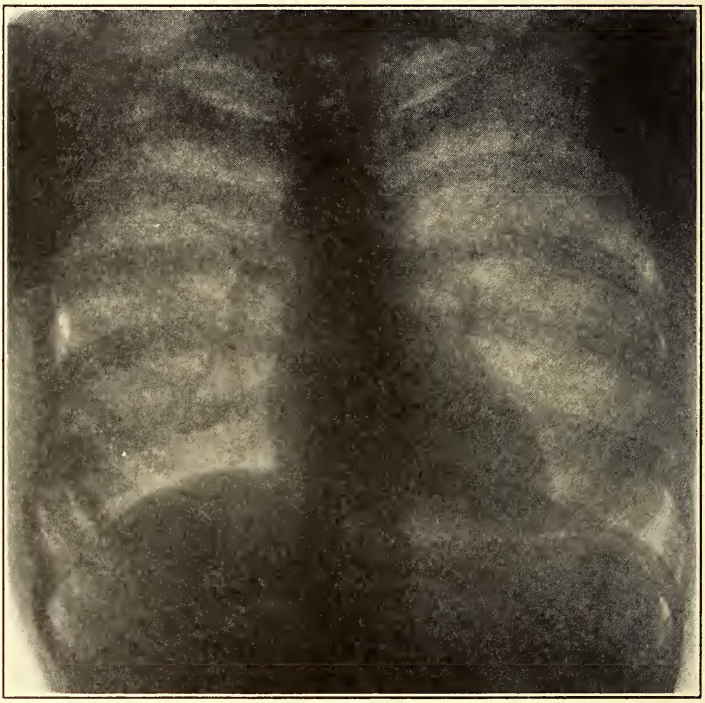

Fig. 4.—Showing the heart in the normal position, with the diaphragm elevated on the right. The enlarged peribronchial glands show well. The case belongs to Class I of the National Association classification.

XXXIV, XXXVII, XL, XLI, XLIII, XLIV, L, LI, LIII, LIV, LVI, LVII, LVIII, LX; 31 cases). With the exception of about 6 cases (Cases XIII, XVIII, XXIII, XXXIV, XLIII, L) all the cases in which enlarged glands were visible were either early or moderately advanced, without the breaking down of tissue; while in the large majority of the advanced cases the glands did not show. It would then appear as if there were two explanations for the absence of glands in the majority of plates in which they were not visible, namely, that their presence was concealed by the area of involvement, or what appears more likely, that with the advance

of the disease the glands had softened or broken down and so failed to give rise to a shadow. There were usually only three or four glands noted in any one plate, and in a number of instances they appeared to be calcified.

Calcification of Costal Cartilages. The presence of calcareous infiltration in the costal cartilages was noted in only 8 cases (Cases I, VIII, XIII, XXVI, XLIV, LII, LVI, LVIII). It would appear to be grossly absent in many cases in which its presence might be expected and in which it could no doubt, be demonstrated microscopically; it was generally noted in the advanced chronic type of the disease, though there were exceptions to this. It was usually confined to the costal cartilage of the first rib, though in one instance it involved them all (Case VIII). In the majority of the advanced cases the involvement was of sufficient density and extent to have concealed the presence of calcification in the cartilages of the first rib, but there was no evidence of calcification in the costal cartilages which could be properly studied.

Summary of Cases. Case I.—F., adult female. Infiltration of upper right lobe. Consolidation of left upper lobe. Lesion more extensive and active on left. Heart normal in position and size. No glands visible. Diaphragm not elevated. Calcareous infiltration costal cartilage first rib. (Class III, N. A.[5])

Case II.—A., adult male. Marked consolidation and fibrosis of upper half right lung, with large cavity in upper lobe. Infiltration of left upper lobe, cavity, with pneumonic consolidation left lower lobe. Heart completely displaced to right. Aorta markedly pulled over. Heart normal in size. No glands visible. Diaphragm elevated on right. Costal cartilages concealed by lesion. (Class III.)

Case III.—H., adult male. Infiltration of right apex and left upper lobe. Heart normal in size and position. No glands visible. Diaphragm normal. No calcification of costal cartilages. (Class II.)

Case IV.—N., adult male. Left-sided hydropneumothorax. Left lung completely collapsed. Disseminated lesions throughout right lung. Complete displacement of heart and aorta to right. Heart normal in size. No glands visible. Diaphragm not visible on left. No calcification. (Class III.)

Case V.—O'C., boy, aged twelve years. Peribronchial infiltration on both sides radiating into apices. Peribronchial glands enlarged. Heart normal in size and position. Diaphragm normal. No calcification. (Class I.)

Case VI.—J., adult female. Small cavity left apex with consolidation (fibroid) of left upper lobe. Slight infiltration right

[5] N. A.—National Association for the Study and Prevention of Tuberculosis.

apex. Heart displaced moderately to left. Aorta also displaced. Heart normal in size. No glands visible. Diaphragm slightly raised on left. No calcification. (Class II.)

Case VII.—J., adult male. Slight infiltration on right. Early case. Heart normal in size and position. Pericardial adhesion at apex. Peribronchial glands enlarged. Diaphragm normal. No calcification. (Class I.)

Case VIII.—J., adult female. Fibroid consolidation both upper lobes, more marked on right. Heart normal size and position. Peribronchial glands enlarged, diaphragm elevated on right. Marked calcification of all costal cartilages. (Class II.)

Case IX.—M., adult male. Large cavity at left apex, with complete fibroid consolidation of rest of lung. Left pleura greatly thickened. Consolidation of right upper lobe. Heart not distinguishable in skiagram, clinically displaced to left. Diaphragm not visible on left. No glands visible No calcification. (Class III.)

Case X.—M., adult, male. Infiltration of both apices. Heart normal size and position. Peribronchial glands enlarged. Diaphragm normal. No calcification. (Class II.)

Case XI.—S., adult, male. Small cavitation right apex, with consolidation right upper lobe. Consolidation left upper lobe. Heart normal in size and position. Pericardial adhesion. No glands visible. Diaphragm normal. No calcification. (Class III.)

Case XII.—A., adult male. Infiltration right apex with marked fibrosis of right lower lobe. Left lung clear. Heart and aorta much displaced to right. Diaphragm much elevated on right. No glands visible. No calcification. (Class II.)

Case XIII.—D., adult, male. Small cavity right apex, with infiltration of right upper lobe. Slight infiltration left apex. Heart normal size and position. Pericardial adhesions at apex. Glands enlarged. Diaphragm normal. Calcification first left costal cartilage. (Class II.)

Case XIV.—R., adult, female. Cavity left apex with consolidation both left lobes. Infiltration right apex. Heart normal size and position. Diaphragm elevated on left. No glands. No calcification. (Class III.)

Case XV.—D., adult, male. Large cavity right upper lobe, consolidation right upper lobe. Small cavity left upper lobe, with infiltration of left upper lobe. Heart displaced to right, normal in size. Diaphragm elevated on right. No glands. No calcification. (Class III.)

Case XVI.—E., adult, male. Infiltration of both apices, more marked on right. Heart normal in size and position. No glands. Diaphragm elevated on right. No calcification. (Class II.)

Case XVII.—V., adult, male. Infiltration both apices, more marked on right. Heart normal, size and position. Glands

enlarged. Heart normal, size and position. Diaphragm normal. No calcification. (Class II.)

Case XVIII.—R., adult, male. Large cavity right apex, another in upper lobe, with marked consolidation of middle lobe. Small cavity left apex with consolidation of left upper lobe. Antero-posterior position of heart. Aorta displaced to right. Pericardial adhesion right side. Glands enlarged. Diaphragm elevated equally both sides. No calcification. (Class III.)

Case XIX.—S., girl, aged seventeen years. Infiltration of right apex, marked emphysema. Heart normal position. Glands enlarged. Diaphragm normal. No calcification. General hypertrophy of heart. (Class I.)

Case XX.—S., adult, female. Infiltration left upper lobe. Heart normal, size and position. Diaphragm normal. Glands enlarged. No calcification. (Class II.)

Case XXI.—V., adult, male. Infiltration roots of both lungs, radiating into apices. Heart normal size and position. Diaphragm normal. Glands enlarged. No calcification. (Class I.)

Case XXII.—D., adult, female. Infiltration right upper lobe. Heart normal size and position. Glands enlarged. Diaphragm elevated on right. No calcification. (Class II.)

Case XXIII.—C., adult, male. Cavities both apices, with consolidation both upper lobes. Marked emphysema. Heart normal in position, enlarged to right. Diaphragm elevated both sides. Glands enlarged. No calcification. (Class III.)

Case XXIV.—Z., adult, male. Cavity left upper lobe. Left-sided localized pneumothorax over partially collapsed lung. Heart normal in size and position. Diaphragm depressed on left. No glands visible. No calcification. (Class III.)

Case XXV.—D., adult, male. Cavity right apex, consolidation right upper lobe. Cavity left apex. Consolidation left upper lobe. Heart and aorta much displaced to right. Heart normal in size. Diaphragm elevated on right. No glands. No calcification. (Class III.)

Case XXVI.—J., adult, female. Infiltration both apices. Tuberculous glands of neck (operative). Heart normal ni size and position. Glands enlarged. Diaphragm normal. Calcification of costal cartilage (left). (Class II.)

Case XXVII.—S., adult, female. Left-sided emphysema (operative) complete collapse of left lung. Heart almost completely displaced to right. Aorta displaced to right. Diaphragm much elevated on left. Glands enlarged. No calcification. (Class III.)

Case XXVIII.—P., boy. Infiltration right apex. Localized empyema on right. Fibrosis right lower lobe and pleura. Heart and aorta not displaced. Diaphragm normal. No calcification. Glands enlarged. (Class III.)

Case XXIX.—P., adult, female. Consolidation left apex. Heart normal size and position. Glands enlarged. Diaphragm normal. No calcification. (Class I.)

Case XXX.—U., adult, female. Infiltration right apex. Heart normal size and position. Glands enlarged. Diaphragm normal. No calcification. (Class I.)

Case XXXI.—H., adult, female. Cavity left apex. Consolidation left upper lobe, disseminated lesions below. Infiltration right apex. Heart slightly displaced to left. Diaphragm elevated on left. No glands. No calcification. (Class III.)

Case XXXII.—B., adult, male. Infiltration both apices. Heart normal size and position. Glands enlarged. Diaphragm normal. No glands. No calcification. (Class II.)

Case XXXIII.—A., adult, female. Large cavity right apex. with consolidation right upper lobe. Infiltration left upper lobe. Heart normal size and position. Aorta much displaced to right. Diaphragm elevated both sides. No glands. No calcification. (Class III.)

Case XXXIV.—C., adult, male. Cavities upper right lobe, with consolidation and marked calcification on right. Cavity left apex with marked consolidation and fibrosis. Heart in anteroposterior position. Diaphragm elevated on both sides. No calcification of costal cartilage. Calcified glands. (Class III.)

Case XXXV.—O., adult, male. Consolidation right upper and middle lobes. Infiltration of left upper lobe. Heart normal size and position. Diaphragm normal. No glands. No calcification. (Class III.)

Case XXXVI.—C., adult, male. Cavity at both apices, with consolidation and fibrosis of both upper lobes. Heart displaced to right. Diaphragm elevated on both sides. No glands. No calcification. (Class III.)

Case XXXVII.—S., adult, male. Infiltration both apices. Heart normal size and position. Glands enlarged. Diaphragm normal. No calcification. (Class II.)

Case XXXVIII.—O., adult, female. Large cavity right apex, consolidation of right upper lobe. Infiltration left upper lobe. Heart displaced to right. Diaphragm elevated on right. No glands. No calcification. (Class III.)

Case XXXIX.—D., adult, male. Large cavity left apex, consolidation left upper lobe. Infiltration right apex. Heart in anteroposterior position. Diaphragm elevated both sides. No glands. No calcification. (Class III.)

Case XL.—McV., adult, male. Infiltration right apex. Heart normal size and position. Glands enlarged. Diaphragm normal. No calcification. (Class I.)

Case XLI.—R., adult, female. Slight infiltration right apex. Heart normal size and position. Glands enlarged. Diaphragm normal. No calcification. (Class I.)

Case XLII.—T., adult, male. Large cavity right apex. Consolidation and fibrosis right upper and middle lobes. Cavity left apex consolidation, left upper lobe. Heart in anteroposterior position. Diaphragm elevated on both sides. No glands visible. No calcification. (Class III.)

Case XLIII.—M., adult, female. Cavities in right upper lobe, with consolidation and fibrosis. Consolidation left upper lobe. Heart displaced to right. Diaphragm elevated on right. Glands enlarged. No calcification visible. (Class III.)

Case XLIV.—McG., adult, male. Infiltration of right apex. Heart normal size and position. Diaphragm normal. Calcification of costal cartilage. Glands enlarged. (Class I.)

Case XLV.—G., adult, male. Large cavity right upper lobe. Consolidation and fibrosis upper and middle lobes. Infiltration left upper lobe. Heart displaced to right. Diaphragm elevated on right. No glands visible. No calcification. (Class III.)

Case XLVI.—L., adult, male. Large cavity in right upper lobe, another in middle lobe. Consolidation of lower lobe, marked fibrosis of right pleura. Consolidation left upper lobe. Heart and aorta much displaced to right. Diaphragm not visible on right. No glands visible. No calcification visible. (Class III.)

Case XLVII.—H., adult, male. Infiltration right apex. Large pleural effusion on left. Heart and aorta much displaced to right. No glands. No calcification. Diaphragm not visible on left. (Class II.)

Case XLVIII.—C., adult, male. Infiltration right apex. Infiltration, with cavity, left upper lobe. Heart normal size and position. Diaphragm normal. No glands. No calcification. (Class III.)

Case XLIX.—P., adult, female. Consolidation upper right lobe with softening. Infiltration left apex. Heart normal size and position. Diaphragm up on right. No glands. No calcification. (Class III.)

Case L.—M., adult, female. Cavity right apex, consolidation right upper lobe with softening. Infiltration left upper lobe. Heart normal size and position. Glands enlarged. Diaphragm elevated on right. No calcification. (Class III.)

Case LI.—M., adult, male. Infiltration right apex. Consolidation left upper lobe. Heart normal size and position. Glands enlarged. Diaphragm normal. No calcification. (Class II.)

Case LII.—A., adult, male. Large cavity right upper lobe, consolidation and marked fibrosis below. Large cavity left apex, consolidation of left upper lobe. Heart completely displaced to right. Aorta markedly displaced. Diaphragm not visible on

right. No glands visible. Calcification of costal cartilages marked. (Class III.)

Case LIII.—G., adult, male. Infiltration both apices. Heart normal size and position. Glands enlarged. Diaphragm elevated on left. No calcification. (Class II.)

Case LIV.—Y., adult, male. Infiltration right apex. Heart normal size and position. Glands enlarged. Diaphragm normal. No calcification. (Class I.)

Case LV.—M., adult, male. Infiltration both apices. Heart normal size and position. Diaphragm normal. No glands. No calcification. (Class II.)

Case LVI.—E., adult, male. Disseminated lesions in both upper lobes, marked emphysema. Heart normal in position, enlarged to right. Glands enlarged. Diaphragm normal. Calcification of costal cartilage. (Class II.)

Case LVII.—C., adult, male. Infiltration of both upper lobes. Heart normal in size and position. Glands enlarged. Diaphragm normal. Slight calcification. (Class II.)

Case LVIII.—M., adult, male. Consolidation right upper lobe. Infiltration left upper lobe. Heart displaced to right. Enlarged to right. Glands enlarged. Slight calcification. Diaphragm elevated on right. (Class II.)

Case LIX.—L., adult, male. Large cavities right upper lobe, with consolidation. Marked fibrosis of lungs and pleura, righ. lower lobe. Cavity left apex, with consolidation upper lobet Heart much displaced to right. Aorta displaced to right. Diaphragm not visible on right. No glands, no calcification visible. (Class III.)

Case LX.—J., adult, female. Infiltration both apices. Heart normal in size and position. Glands enlarged. Diaphragm normal. No calcification. (Class II.)

REVIEWS.

THE PRINCIPLES OF PATHOLOGY. Vol. I. *General Pathology.* By J. GEORGE ADAMI, M.D., LL.D., F.R.S., Professor of Pathology in McGill University, Montreal. Pp. 948; 322 engravings and 16 plates. Vol. II. *Systemic Pathology.* By J. GEORGE ADAMI and ALBERT G. NICHOLLS, M.A., M.D., D.Sc., F.R.S. (Can.), Assistant Professor of Pathology and Lecturer in Clinical Medicine in McGill University, Montreal. Pp. 1082; 310 engravings and 15 plates. Philadelphia and New York: Lea & Febiger, 1909.

PROFESSOR ADAMI'S *Pathology* has from the date of its issue established a new standard for similar publications in America; and has lifted whatever opprobrium may have been fancied in the often repeated remark that there has been no American pathology (in the sense, of course, that the text-books of this hemisphere have followed more or less closely the thought, plan, and substance of European, notably German, authorities).

The first of these volumes, now in the second year after publication, has been widely studied; and expectancy has changed generally to cordial admiration for the breadth of fundamental discussion, the mode of presentation, and the clearness of expression of established knowledge, as well as for many of the personal views and applications introduced by the author. There have been numbers of excellent text-books issued from American presses which have not failed in matter of systematization of the subjects and in description of pathological processes and lesions; but in the endeavor to set cause to effect, to elucidate the rationale of events, and to explain the eternal "how and why" in the study of disease, the author's breadth of training and viewpoint, as well as his experience in long years of teaching, have combined to make the work notable.

A large part of this first volume is essentially preliminary, devoted to introductory consideration of the cell as a unit of vital organization, discussing the details of cellular structure and interrelation in complex organisms, our knowledge of the chemistry and physics of cellular activity, growth, multiplication, adaptation, and differentiation, and the data and problems of reproduction and inheritance. The essence of these chapters lies in the author's conception of the cellular protein molecules as elemental structures, biophores, the

various phenomena of energy being referred to changes in these; the familiar side-chain theory being applied in explanation of their constitution and their changes in metabolism. This same idea is followed in the presentation of his views of cellular growth and differentiation, of adaptation, variation, and evolution, as well as the phenomena of inheritance. The more recent publication of Reichert and Brown upon the hemoglobins of the animal kingdom, while not directly related, will be found to lend considerable confirmation to Adami's view, in that the essence of distinction in evolution, and probably, too, in all vital phenomena, whether normal or pathological, must be carried back to molecular constitution. Such views are rapidly permeating our newer conceptions of biology, since the development of physical chemistry; and are bound in the near future to dominate medicine, just as in the past, one after another, the cellular pathology and germ theory of disease bore in upon us. That new cells developing from the original fertilized ovum grow by side-chain accretions to their molecules; come to differ, as slight differences in pabulum in diverse locations obtain; and progressively diverge as the diverging products modify more and more the side-chain construction of appropriating molecules, until the complete cellular differentiation of the body, with the harmonious interdependence and mutual resistance of its cells, is established—this is basic. The protoplasmic molecule of one cell is in its general structure like the protoplasm of a cell of a different organ, or like a cell of the same part from a different animal; and the differentiation in the individual, or the evolutional difference in different species, lies mainly in the side-chains, in their different qualities, valences, and affinities. The chromosomic theory of inheritance has never been entirely satisfying; but one can with Adami see under it in the possibilities of the chemical interaction of the complex biophores of the germ cells, a rational explanation for the dominance of one parental type, the chance for variations and mutations, and can see a reason for the only certain inheritance of acquired characteristics of the parents we know, that which follows those constitutional and toxic influences which may fixedly modify the molecular constitution of the germ cells, and in turn the progeny of these modified cells.

Thereafter, after discussion of antenatal acquirement, so often confused with true inheritance, the author devotes the remainder of the first part of the volume to the causes of postnatal acquirement of disease and the pathological processes which may logically be regarded as directly reactive or responsive to these—inflammation as a local reaction, infection with its general response in pyrexia and other phenomena, and the reactive immunity induced, as well as syncope, shock, and collapse as typifying failure of or negative reaction (death, however, being left to a subsequent section in the latter part of the volume). Of the chapters on pathogenic influences, including those of mechanical, physical, chemical, and parasitic

natures, the more notable are devoted to the endogenous intoxications from internal secretory faults and faults of metabolism and to the effects of overstrain in structural and physiological sense, and to cellular disuse.

The chapters on inflammation, after the author's well-known plan of considering the subject in a comparative manner in the simpler organisms leading up to vertebrates in order to fix the essential features of the adaptive reaction, might well stand as a type of the methods pursued throughout the volume. Whatever the injury (and the author takes the safe ground that it is by no means always of microbic origin), the two prominent factors in the process are the proliferation of the cells about the injured area and the attraction of the wandering cells to the area, the role of the bloodvessels being strictly secondary in that it really but facilitates the former. One could suggest that with these basic factors more stress might be laid upon the entrance of excess of the body fluid into the area with its general and special influences toward removal of the cause of the process; and, too, many may miss in this luminous discussion a definitive presentation of the resolution of inflammation, aside from the matter of repair and the fate of the leukocytes and fibrin, for there are additional problems in the absorption of exudate and liquid waste, in the resumption of vascular tone and similar features. Ehrlich's side-chain theory is basic to the disquisition upon immunity; but the author is not bound rigidly to an immediate and essential chemism between the antigen and antibodies, realizing the possibilities of physical relations entering into the problems afforded by the recognized phenomena and well brought forward in the later trend of study.

In the second part of the volume the familiar progressive and regressive pathological changes of less definite relation or of unknown relation to cause are presented, the author breaking away from the common habit of introducing here, however, the hyperemias, ischemia, hemorrhage, thrombosis, embolism, and œdema, reserving these for the second volume in connection with the circulatory system. The line of discussion includes in the first group hypertrophy, regeneration, transplantation, metaplasia and the neoplasms; in the second, the atrophies, abioplasia, reversions, degenerations and infiltrations, necroses, and somatic death. In the descriptive part of each clearness and sufficiency, as may be expected, prevail; but, as in the first part of the volume, the notable features lie in the analysis of cause and relation and in the presentation, where this is impossible, of reasonable working theory. It is an open question as to the value of adding to existing morphological or relative classifications of the tumors; if it be granted, there are points of excellence in the author's separation of neoplasms into those of the lining (lepidoma) and those of the pulp tissues (hyloma), although it removes none of the difficulties in routine employment of Cohnheim's basic arrangement.

Whatever the cause of a tumor, Adami would hold there is assumed a peculiarity in its elements, not so much shown morphologically as in the predominance of a vegetative over the ordinary functionating character of the cell. Why such a character is assumed may be a matter of hypothesis, but there is reason to suppose that just as mutations in animals and plants are not mere chance, but determined more or less by alterations in environment, so it may be thought possible that cellular mutations of the type in question may by a variety of internal somatic conditions be determined, and there is no reason that stimuli of external origin, bacterial, chemical, or physical, may not do the same. He would look to no specific cause for tumors and seek for no parasite as definitely *neoformans*. The tumor cell itself is a modified body cell, and in its modification is the specific element (itself the antigen) and working out, the more atypical it is, its own antibodies from the somatic reaction to itself. Gaylord's work showing the development of immunizing substances in mice recovering from certain tumors, that of Jensen in inducing disappearance of tumor growth in mice by injecting into the animals the elements of a part of the tumor itself, as well as that of Coca along similar lines, and the recent announcement by Hodenpyl of a curative material for human cancer shown in the ascitic fluid of a cancerous human being, and other work of the same type, are all leading to a similar conclusion; and it is a safe prediction by the author that ultimate triumph over these growths is far from hopeless.

In the second volume, Prof. A. G. Nicholls collaborates with Adami. The association is valuable, no doubt, in a number of ways, but at the same time it leads to occasional lack of perfect harmony between the products of the two authors, the senior writer commonly presenting for each section an introductory portion dealing with the broad pathological problems in structure and physiological relation, and the junior author assuming the details of gross and minute anatomical description. Systemic pathology for its greater attractiveness should be presented in as fully applied form as possible, with frequent indication of the relation of the existing anatomical lesion with the symptoms manifested by the living subject, and with the distinct purpose of correlating with the primary lesions the secondary and complicating faults which invariably arise and, as a rule, prove in their combination the cause of death rather than the isolated primary lesion. Herein the authors are hampered by lack of space; and the fault in mind is not a qualitative but rather a quantitative one. It may be remedied in future editions by fuller discussion of the functional effects of at least the more important types of lesions, giving to classes of students a more certain habit of reasoning from a pathological basis in their clinical studies and at the same time insuring a more ready application of pathological knowledge by the practitioner. There is little reason for an

elaborate description of this second volume, which follows through the diseases of the blood, cardiovascular and hemopoietic organs, respiratory and other systems of the body in regular order, each with excellent anatomical exposition of its important lesions and with sections upon the broader pathological physiology of each, which are as valuable from the infrequency of such discussions in works on pathology as from their intrinsic excellence.

If, as Professor Adami says in his preface, the book was twelve years in its forming, it is worth all the time and the effort. It cannot, of course, remain indefinitely fresh: there is too rapid progress for such expectation. But it has been shaped along lines which are permanent or at least look far into the future; and is certain for its many excellences to be long-lived by repeated revision without actual recasting. A. J. S.

A Text Book of Physiological Chemistry for Students of Medicine. By John H. Long, M.S., Sc.D. Second edition. Philadelphia: P. Blakiston's Son & Co., 1909.

The subject matter of Dr. Long's book is divided into four sections: I. The Nutrients; II. Ferments and Digestive Processes; III. The Chemistry of the Blood, the Tissues, and Secretions of the Body, and IV. The End Products of Metabolism. In this edition a few changes have been made, notably the adoption of the protein classification recommended by American biological chemists, and a new chapter on the methods used in urine analysis.

One is rather appalled at the outset by being confronted with a sentence containing 134 words, of such involved construction and of such obscure meaning that if asked what he read, one might well reply with Hamlet, "Words, words, words!" The entire book while not guilty again of such verbosity, is nevertheless quite beyond the understanding of the medical student. Unless he is exceptionally well grounded in organic chemistry the book in many parts is incomprehensible, and the undergraduate who can read Chapters III and IV with an intelligent grasp of the subject is the fortunate possessor of unusual training and intellect. Chapter XIV, dealing with the complicated theme of special properties of blood serum, reflects great credit on the author. Dr. Long has presented this subject in a clear, concise, and easily understood manner, and the only adverse criticism that might be raised is that the author would have made his topic more clear had diagrammatic illustrations been shown. The chapter devoted to urinary examinations is hardly complete enough in detail for one to make even practical metabolic studies, and the index is very unsatisfactory. As a text-book the work is too lacking in explanation, and we fear the student will be unnecessarily confused by the at best recondite subject. As a laboratory book used in conjunction with practical demonstrations and intelligent instruction it may find a field of usefulness. E. H. G.

Chemical and Microscopical Diagnosis. By Francis Carter Wood, M.D., Professor of Chemical Pathology in the College of Physicians and Surgeons, Columbia University, New York. Second edition; pp. 725; 192 illustrations. New York and London: D. Appleton & Company, 1909.

This book, which comprises about the best of our knowledge on the subject, is a noteworthy addition to laboratory literature. It describes, for the most part, in good working detail, the examination of the blood, gastric contents, feces, parasites, oral and nasal secretions, sputum, urine, transudates and exudates, and milk. Some of the methods as given are lacking in essential points of description, and certain well-known tests have failed to find a place in the work under discussion. The author has chosen to give the reference where a new method was first published, and this would seem advisable in all laboratory manuals, since it is apparently impossible to find accurate record of technique in books of this nature. The plates and illustrations are uniformly good, and it is a rather novel experience to make new acquaintances in the pictorial line, instead of meeting, as has been the reviewer's misfortune in the past, one's old friends reproduced in book after book. Dr. Wood's second edition is to be heartily recommended; those engaged in laboratory practice will find it a most useful addition to an already long list of laboratory books. E. H. G.

Lehrbuch der klinischen Diagnostik innerer Krankheiten. Edited by Paul Krause, M.D., Professor and Director of the Medical Polyclinic in Bonn, Germany. Pp. 922; 360 illustrations. Jena: Gustav Fischer, 1909.

The *Text-book of the Clinical Diagnosis of Internal Diseases*, edited by Professor Krause is the composite work of thirteen collaborators. Professor Krause himself contributes chapters on the methods of examining patients, on *x*-ray examinations, and on clinical bacteriology; Professor Wandel, of Kiel, discusses the anamnesis and the general habitus of the patient, and the diagnosis of the acute infectious disease; Professor Lommel, of Jena, diseases of the upper air passages and exploratory puncture and cytology; Professor Gerhardt, of Basle, diseases of the respiratory apparatus; Professor Staehelin, of Berlin, diseases of metabolism, and in association with Professor Ortner, of Innsbruck, diseases of the circulatory apparatus; Professor Winternitz, of Halle, diseases of the urogenital tract; Professor Ziegler, of Breslau, diseases of the blood; Professor Mohr, of Halle, diseases of the digestive tract; Professor Jamin, of Erlangen, and Professor Finklenburg, of Bonn, diseases of the

nervous system; Professor Hertel, of Jena, diseases of the eye in internal diseases; and Professor Esser, of Bonn, diseases of infants. The book is well written, and sufficiently comprehensive, since although it includes about all that is necessary there is little if any mention of etiological factors and of matters of doubtful moment. It may be said to be representative of the present German school of medicine, and as such is to be highly commended. A. K.

Cataract Extraction. By H. Herbert, F.R.C.S., Late Lieutenant-Colonel, I.M.S., Professor of Ophthalmic Medicine and Surgery in the Grant Medical College, and in charge of the Sir Cowasjee Jehangir Ophthalmic Hospital, Bombay. Pp. 391. New York: William Wood & Co., 1908.

This work is equally valuable for its abundant citations from the writings of others who have treated of the same subject and for the fruitful lessons the author has drawn from his own experience, comprising as it does about 5000 extractions; and even this number he declares to be small compared with the work of other ophthalmic surgeons in India. The writer tells us that grave conjunctival disease is much more common in India than in Europe or America. This unfavorable condition has to be dealt with speedily and efficiently; abundant douching with bichloride solution, 1 to 3000, is the mainstay and has yielded the most satisfactory results—indeed, so satisfactory that evil is turned to good; the douchings being rarely necessary in the western world, they are omitted in the occasional cases where they would prevent infection. Of 1655 extractions, not a single suppuration occurred, certainly justifying the author's claim of a near approach to perfection in this respect. We confess to some surprise, however, at the statement that nasal infection through the lacrimal passages does not take place. The chapter descriptive of the operation, which takes up nearly one-half of the whole book, is very thorough even to minuteness. The combined operation is considered to be the standard. The capsule is divided vertically with the cystitome and the delivery of the lens is aided by fixation forceps, differently applied in accordance with special indications. Irrigation is employed when necessary to remove blood, etc. Chapter IV deals with "variations in procedure," the most valuable portion of which is the critical appreciation of the merits and faults of methods other than the writer's. There is necessarily considerable repetition here, but this is hardly a fault for the serious student.

In a work so meritorious as this one, for which the entire ophthalmic world will be sincerely grateful, it seems ungracious to seek

out any shortcomings. While we rise from perusal of the book instructed as from no other with which we are acquainted upon the subject of modern methods for operating upon cataract, we have a feeling that the subject is presented somewhat obscurely and that the reader fails to get as clear an idea of the whole as the excellent matter deserves. A little greater attention to method will easily overcome what is a fault of form but not of substance. T. B. S.

A Text-book of Diseases of the Ear. By Macleod Yearsley, F.R.C.S., Senior Surgeon to the Royal Ear Hospital, London. Pp. 452. Chicago Medical Book Co., Chicago, Ill., 1909.

This book is, as is stated in the preface, an expansion of a previous work of the author on *Common Diseases of the Ear,* but it is really an entirely new publication, and in its present form is justly entitled to rank as a very complete text-book of otology. In the arrangement, it follows the customary classification of the various subjects, although there are two useful chapters included on somewhat unusual lines, namely, Chapter XII, on the "The Influence of General Diseases of the Ear," and Chapter XV on "The Medico-legal and Life Assurance Aspects of Otology." The book is thoroughly up to date in its consideration of all the most recent developments in the science of otology. There is an excellent, though brief, account of the recent advances in our knowledge of the physiological and pathological conditions of the labyrinth. The various operations upon the temporal bone are well described, and the subject of the intracranial complications of aural disease is excellently considered. Like most English otologists, the author adopts Lake's classification of the results of tests for bone and air conduction by Rinné's method, using Greek letters as symbols for the test. To most American aurists, such a classification simply serves to complicate, and, as a rule, they prefer writing out the test result in full, to the use of an arbitrary symbol. The illustrations throughout the book are generally original, and of most excellent quality. It can be safely commended to the student of otology as an excellent epitome of the subject. F. R. P.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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The Effect of Digitalis on the Ventricular Rate in Man.—Of the cardiac irregularities produced experimentally by digitalis, the earliest to appear is usually an occasional omission of ventricular contractions, owing to the blocking of the stimulus from auricle to ventricles. A somewhat late phenomenon is the production of a complete auriculoventricular dissociation which differs from ordinary heart-block in that the ventricular rate is not slow, but approaches, and usually exceeds that of the auricles. Although a common result of digitalis poisoning in dogs, this condition has never been noted in man except in the case reported by HEWLETT and BARRINGER (*Arch. Int. Med.*, 1910, v, 93). Their patient, a man, aged twenty-seven years, with chronic myocardial insufficiency, who had taken digitalis in moderate doses over a considerable length of time, developed on the day before his death, a remarkable condition. Tracings of the venous pulse and apex showed a regularly recurring cycle of changes apparently depending on the interference of two systems of waves which were independent of each other, and not quite synchronous. Each cycle lasted about seven seconds and included fourteen ventricular contractions. The two systems of waves were evidently due to the auricular and ventricular contractions, and the rates were such that for thirteen auricular there were fourteen ventricular contractions. Hewlett and Barringer believe this to be the result of a cumulative action of the digitalis, and call attention to the fact that it may be difficult to ascertain when enough of the drug has been given, for at no time was there a slowing of the pulse. While in experimental heart-block the rate of the ventricle is increased by digitalis, there is little clinical evidence on the subject. In a case of complete heart-

block with slow pulse, however, the same writers failed to note any increase of ventricular rate after the use of moderately large doses of digitalis. It is possible that the appearance of extrasystoles and the temporary disappearance of the *a* waves from the jugular pulse (due to a toxic weakening of the auricular contractions?) may have been due to the drug.

Auricular Fibrillation.—It is well known that in the latest stages of cardiovascular degeneration, especially in mitral stenosis, the pulse often becomes exceedingly irregular, and in the jugular the wave of auricular contraction disappears. This has long been regarded, particularly by Mackenzie, as depending upon the origin of the rhythm at the node of Tawara (hence the term nodal rhythm). Lewis (*Brit. Med. Jour.*, 1909, ii) asserts that facts are at his disposal permitting the conclusion that the rhythm arising in the neighborhood of node gives rise to a different clinical picture. This conclusion is based upon the study of an instance of paroxysmal tachycardia in which auricle and ventricle contract together. Secondly, the pulsus irregularis perpetuus is dependent upon fibrillation of the auricle. This conclusion is based upon the fact that the rhythm is exactly similar to that which may be produced experimentally by inducing fibrillation of the auricle, and is a unique condition. Lewis points to the fact that electrocardiograms taken from patients exhibiting this irregularity, show a number of irregular waves apart from the ventricular curve, and more clearly defined in diastole. Such waves are found in no other disorder of the heart action. They disappear when irregularity vanishes, are not evident upon the cardiogram, and are identical with the curves yielded by fibrillation of the auricle. Furthermore, synchronous tracings show that the waves in the experimental cardiogram correspond to the fibrillary movements of the auricle. [In connection with this interesting communication it may be remembered that Cushny and Edwards in the American Journal of the Medical Sciences, 1907, cxxxiii, 66, arrive at the conclusion that an instance of paroxysmal irregularity was probably due to this cause.—W. S. T.]

The Etiology of Beri-beri.—The studies of Fraser and Stanton at the Institute for Medical Research, Federated Malay States (*Trans. Soc. Tropical Med. and Hygiene*, 1910, iii, 257), are based on the chemical analyses of various types of rice, and on the production of polyneuritis gallinarum, a disease analogous to beri-beri, by feeding experiments in fowls. It was first found that Siam rice, which is most often associated with epidemics of beri-beri, contains a lower percentage of fat, than either Rangoon rice or parboiled rice. Microscopic sections showed that in Siam rice the pericarp, the outer layer, containing most of the aleurone and oily material, had been removed by the process of polishing. The relation of the milling of rice to the production of the disease in fowls was then studied. Fowls fed on the original padi ale remained healthy. Of twelve fowls fed on the finished, polished rice, six developed polyneuritis. Other fowls fed on the same finished rice, plus the polishings, all remained healthy. From these experiments Fraser and Stanton concluded that the polishing of white rice removes from the seed some substance essential to the maintenance of the

normal nutrition of nerve tissues. It was further shown that staleness of rice, or the development in it of poisonous substances subsequent to its being milled are not important factors. Parboiled rice, in itself healthy, when extracted with alcohol, caused polyneuritis in fowls, but the addition of the alcoholic extract to a rice known to be injurious prevented this disease. Further chemical investigations showed that the power of a rice to produce polyneuritis gallinarum varied with its phosphorus content—the higher the phosphorus content, the less liable was it to be injurious. The highest percentage of phosphorus was found to be present in rice polishings. Moreover, the addition to an injurious rice of a quantity of polishings which contained enough phosphorus to bring the total phosphorus content up to that of parboiled rice sufficed to preserve nutritive equilibrium. The prevention of beri-beri thus depends on the substitution of ordinary white rice, by a rice in which the polishing process has been omitted, or carried out to a minimal extent, or by the addition to a white rice diet of articles rich in those substances which are not present in sufficient amount in white rice. One such article, which is cheap, and may be readily obtained, is the polishings from white rice.

The Physiology of the Immediate Reaction of Anaphylaxis.—On the injection of a dose of horse serum into the vein of a previously highly sensitive guinea-pig, there occurs a chain of symptoms—chiefly of respiratory nature, which result in the death of the animal in from three to five minutes. While the general type of the reaction has been reported and confirmed by various observers, it has remained for Auer and Lewis (*Jour. Exper. Med.*, 1910, xii, 151) to study and explain the physiological basis. While convulsive and paralytic symptoms may dominate the picture if the animal is loose, they found that if it is held in a suitable holder these are less marked and the respiratory changes come into the foreground. They thus paid especial attention to the lungs, and found, as did Gay and Southard, that at autopsy the lung in acute anaphylaxis tends to remain in an inspiratory, distended condition with open thorax, with unobstructed trachea, and large bronchi, and without obvious pulmonary œdema. This immobilization of the lungs they consider to be the most characteristic sign of immediate anaphylaxis in the guinea-pig. Experiments were performed in which the respiratory movements of the chest and the volume changes of the pleural cavity were recorded, as well as others in which the animals were allowed to breathe from a bottle, and changes during inspiration and expiration registered. As a result it was evident that some stenosis is gradually produced in the pulmonary passages so that in the final stage practically no air enters or leaves the lung in spite of violent respiratory attempts. Death is due to asphyxia. The characteristic reaction of anaphylaxis was also obtained in pithed animals, showing that its production depends on a peripheral process in the lung, and not on the central nervous system. After reviewing the possible cause of the condition, the authors conclude that it is due to a tetanic contraction of the muscles of the finer bronchioles, so that air is imprisoned in the areolar sacs. Atropine, which paralyzes the bronchial muscles, may, under certain conditions, be able to relax the anaphylactic lung so that it is again able to expand and contract. The blood pressure

in immediate anaphylaxis first shows a considerable rise, and then a gradual drop to 10 to 20 mm. Shortly after the injection of the toxic dose, a heart block, often with a 3 to 1 rhythm, develops, and is probably due to asphyxia. The cardiac vagus gradually loses its irritability after injection of the toxic dose.

Jaundice in Pneumonia.—As a result of the experimental study of cholecystitis, LEMIERRE and ABRAMI have previously shown the important part played by descending, hematogeneous infections in the production of inflammation of the bile passages. They now (*La presse méd.*, 1910, No. 10, 82) report three fatal cases of pneumonia associated with jaundice in which bacteriological examination of the bile at autopsy showed pure cultures of pneumococcus. In all three instances the stools were colorless, but careful search revealed no obstruction in the bile passages. The fluid in the gall-bladder was nearly colorless, and in two of the cases failed to give a Gmelin reaction. They believe that the primary cause of the jaundice is an involvement of the liver parenchyma, and that any inflammation of the bile ducts is purely secondary. In all of the cases there were signs of alcoholic cirrhosis of the liver, and Lemierre and Abrami consider that hepatitis complicating pneumonia is rare except when the liver has been the seat of some previous pathological process.

On the Quantity of Glycuronic Acid in the Urine in Health and Disease.—TOLLENS and STERN (*Hoppe-Seyler's Ztschr. f. physiol. Chemie*, 1910, lxiv, 39) have found, by means of a new quantitative method recently described by Tollens, that the excretion of glycuronic acid in the urine is far greater than has generally been supposed. Mayer and Neuberg, for example, give the daily average output as 0.004 gm. per 100 c.c., whereas the authors find 0.025 gm. per 100 c.c. or 0.3 to 0.4 gm. in the twenty-four hours' urine as the average in health. In several cases of diabetic coma, they have encountered a complete absence of glycuronates in the urine, tested with the naphthoresorcin test. Administration of sodium salicylate, which causes a marked augmentation of the glycuronates in the urine as a rule, failed to produce a positive reaction in these cases. Various drugs, especially salicylates and chloral hydrate, combine with glycuronic acid in the body; after their administration, the glycuronic acid may amount to 1.4 gm. per diem. In one case of carbolic acid poisoning (25 gm.? taken) the urine, blackish green in color and definitely levorotatory, contained 8.5 gm. of glycuronic acid on the first day. Such a urine, of course, is capable of reducing Fehling's solution.

The Cultivation of the Organism of Infantile Paralysis.—It is of interest in relation to the recent publications of Flexner and Lewis on the etiological factor of anterior poliomyelitis, to receive the results reported by LEVADITI (*Presse médicale*, 1910, No. 6, 44) in a preliminary note from the Pasteur Institute. In several experiments he has inoculated bouillon to which the blood serum of monkeys and rabbits has been added, with active filtrates containing the specific organism. In one instance the medium became cloudy after being kept in the themostat for ten days. At the end of fifteen days the culture was injected into a monkey. After

an incubation period of twenty days paralysis set in, thus demonstrating that the organism retains its virulence for at least fifteen days at 38 degrees. Microscopic examination of the cloudy medium by ordinary methods showed no micro-organisms, but after centrifuging, dissolving the clot and mordanting after fixation by alcohol or heat, he was able to find a large number of round or rather oval bodies, appearing in pairs or in masses. They are extremely small and are sometimes polymorphous. They do not stain well with aniline dyes, but after prolonged staining with dilate fuchsin assume a pale pink color, or appear as clear dots surrounding by a pinkish zone. Control experiments with culture media which had not been inoculated showed only granules of quite different size and shape.

Rat-bite Fever.—Horder (*Quarterly Jour. of Med.*, 1910, iii, 121) has collected three instances of an apparently specific kind of blood-poisoning following the bite of a rat. The most prominent symptom noted was periodic fever beginning from twenty-one to twenty-eight days after the occurrence of the bite. The temperature rose to 103° to 104°, fell to normal in two to three days, and then rose again in the course of the next few days. In one case the remission continued over several months. During the febrile periods there was a well marked leukocytosis. In two instances there was a blotchy erythema, and in one of these indurated plaques and diffuse tender subcutaneous nodules were present. Blood cultures and the inoculation of blood into animals gave no results and the examination of stained specimens of blood failed to reveal any parasites. There was no enlargement of the spleen. The prolonged incubation period, the form of the fever and the absence of suppuration in the original wound make it unlikely that the cause of the disease is a pyogenic infection secondary to the bite. Horder considers that the etiological factor is probably a protozoon. [It is interesting to note that Quincke (*Mitt. aus. d. Grenzgebeit, d. med. u. Chir.*, 1900, v, 231) has reported eleven cases of almost exactly the same symptom-complex which occurred in Japan. He also refers to a number of articles in the Japanese literature, and states that while there is no mention of it in European literature, a characteristic remittent febrile disease following the bite of a rat after a more or less prolonged incubation period, has been recognized in Japan for many years.—W. S. T.]

"Nail-palpation" of the Arterial Wall.—Wertheim-Salomonson (*Deut. Arch. f. klin. Med.*, 1910, xcviii, 596) calls attention to the well-known difficulties of palpating the arterial wall (1) where the arterial wall is thin, (2) when it is densely covered with fat, and (3) when the blood pressure is high. He proposes a method, which he calls "nail-palpation" (Nagelpalpation), by means of which any arterial wall, whether it can be palpated in the usual way or not, may be felt, and its thickness fairly well judged. Instead of palpating the artery with the ball of the finger, the finger-nail is used. The nail is placed perpendicularly on the surface of the skin, so that the edge of the nail runs parallel with the long axis of the artery. By moving the finger transversely across the artery, the latter slips under the nail as if it were dissected out. The artery may be palpated in this way almost as readily in young children as in adults. With a little practice, Wertheim-

Salomonson says one quickly learns to recognize arterial thickening. The method is applicable to the palpation of any artery which rests on a firm bed, as well as to the palpation of many superficial nerves.

A Previously Undescribed Symptom of Tetany.—H. SCHLESINGER (*Wien. klin. Woch.*, 1910, xxiii, 315) has observed a new sign in a typical case of tetany, which he designates the "Beinphänomen." The sign is elicited in the following manner: If one seizes the leg with the knee joint extended and then flexes the thigh on the abdomen, in a short time (at the most, two minutes) an extensor cramp develops in the knee with extreme supination of the foot. The phenomenon may appear when the patient sits up in bed. If the trunk is flexed on the thighs, the spasm likewise appears. Like the Trousseau phenomenon, the new sign can be brought out in the intervals between attacks. The frequency of occurrence of the Beinphänomen in tetany cannot be foretold as yet; nor is it known that the sign is one peculiar to tetany.

SURGERY.

UNDER THE CHARGE OF

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Atlo-axoid Fracture Dislocation.—PILCHER (*Annals of Surgery*, 1910, li, 208) reports a case which has been under his care for a period of nearly ten years, and therefore represents the possibilities of ultimate repair and restoration of function which may take place in such cases. The patient, a man, aged thirty-three years, fell headlong a distance of fifteen feet, striking on his forehead. His head was notably bent over toward his left shoulder and was fixed in such great flexion that he could not open his mouth more than a half-inch. He had no symptoms other than suboccipital pain and the deformity and stiffening of the neck. During the following two months he was conscious of a growing lack of power in his lower limbs, most marked on the right side. Shortly afterward it was found that he had no power in his right arm or leg, except a little in the fingers and toes. Catheterization became necessary. Pilcher saw him first about three months after the injury, and six weeks later the atlas and axis were exposed by operation. A forward dislocation of the atlas upon the axis was demonstrated, but careful attempts to reduce it were futile and the wound was closed. A slight improvement developed in the subsequent weeks. He became able to empty his bladder spontaneously and two months after the operation

began to sit up. He was still hemiplegic when he left the hospital ten weeks after the operation. A gradual return of power in the paralyzed leg manifested itself after his return home and continued until the normal condition returned. Less improvement occurred in the right upper extremity. Nine years after the accident the deformity of the head and neck and the immobility were unchanged. He is able to walk normally fairly long distances without fatigue. There is no differences in strength between the two lower limbs. The bladder function is normal and his mentality is unaffected.

Malignant Degeneration of Benign Diseases of the Breast.—Speese (*Annals of Surgery*, 1910, li, 212) follows Warren's classification. He finds that certain tumors which present symptoms of malignancy do not show malignant histological changes, and, on the other hand, carcinoma occasionally arises in a preëxisting tumor without causing symptoms indicative of such a transformation. It is conlcuded that operative interference in all tumors of the fibroepithelial type, is indicated to prevent this complication. Two instances of carcinomatous changes were found in 17 cases of periductal fibroma studied pathologically. In both there were one or two symptoms which were only suggestive of cancer. Abnormal involution (chronic mastitis) occurs more frequently than any other affection of the breast with the exception of carcinoma. In 180 cases of breast disease, Speese found it in 18 per cent., and of the 35 cases studied in the laboratory, 9 instances of malignancy were encountered (26 per cent.). In 295 cases of abnormal involution reported by 9 different writers 44 were found to be carcinomatous (15 per cent.). In doubtful cases exploratory incision is indicated; a careful search throughout the entire part involved is necessary, for the malignant area is apt to be small. Malignancy being detected, a radical operation should be performed. The exploratory incision does not reduce in any way the chance of ultimate cure, whereas exploratory incision followed by the radical operation for malignancy at a later period has been invariably fatal according to Bloodgood. The bilateral character of the disease is one of the interesting features and one for which occasional double amputation has to be performed. Cystadenomas, cancer cysts and mastitis are also discussed. Areas of induration following mastitis should receive as careful attention as other forms of benign disease, early removal of which may remove its greater danger.

The Treatment of Cystitis, Especially, Severe Postoperative Cases.—Schlafi (*Zeit. f. Gyn. u. Urol.*, 1910, ii, 4) says that in most cases he has not used irrigations but has depended chiefly upon internal therapy, with flushing and disinfection from within. For urinary antiseptics he has employed aspirin, benzosalin, novaspirin, and diplosal. Of these the most effective was aspirin. When irrigations were employed the quantity injected was never so great that it distended and irritated the bladder walls. It was given slowly and regularly and the temperature of the fluid was usually between 18° and 20° C. Only somewhat persistent acute and subacute catarrhal conditions and especially severe pus cases, make irrigation necessary. Aniodol was found to be the best antiseptic solution for the irrigation. It is a formaldehyde preparation with some sulphozyanallyl. Its bactericidal properties are greater than

that of other urinary antiseptics. According to Fouard's investigations it can be said that it gives the greatest therapeutic effect with the least danger, since it is neither caustic nor toxic like sublimate and carbolic acid. Schlafi used it in a 0.25 per cent. solution, which gave it a sufficient concentration and produced no symptoms of irritation. It has simplified the treatment of cystitis. It provides for the mechanical cleansing, and the removal of the decomposed urine and its contained pus; and it provides the best bactericidal effect without the disadvantages of other equally strong disinfectants.

The Operative Treatment of Wounds of the Lungs.—Möller (*Archiv f. klin. Chir.*, 1909, xci, 295) says that up to a few years ago penetrating wounds of the thorax involving the lungs, were generally treated conservatively, with rest in bed, morphine, ice, and antiseptic treatment of the wound. A simple occlusive dressing, or incision and tampon of the wound in the thoracic wall or suture of the wound was employed. For some years efforts have been made to find the lung wound, through a sufficient opening in the chest wall, and to suture or tampon the wounded lung surface. Stucky reported 25 cases of wounds of the lungs treated by suture, and concluded that in every stab wound of the thorax coming into the hospital within twenty-four hours after the accident, the ribs should be resected, the lung wound exposed and sutured. This led to the collection and study of similar cases from Körte's clinic, from which it was determined that the radical operation proposed by Stucky, not only was not necessary, but was improper; and that these wounds healed with simple occlusive dressing. In some few severe cases free exposure and direct treatment of the lung were justified. The material studied consisted of 90 cases in which the pleura and perhaps the lung were wounded, 48 by gun-shot, 19 by stab or incision, and in 23 there was a subcutaneous laceration of the lung, 12 with and 11 without a fracture of the ribs or sternum. Of the 48 gun-shot cases, the symptoms occurred as follows: Hemothorax, 37 times; hemoptysis, 21 times; pneumothorax, 12 times; and connective tissue emphysema, 9 times. The treatment and course were as follows: Puncture and aspiration, 10 times; empyema, 4 times; rib resection, twice; excision of the shot, 14 times. Death resulted in 7, and the average duration of healing was five to five and one-half weeks. The following complications occurred: Wound of the pericardium in 5, of the heart in 2, of the diaphragm and abdominal organs in 2, and of the spinal canal in 1. Of the 19 stab wound cases, the prominent symptoms occurred as follows: Hemothorax in 9, hemoptysis in 3, pneumothorax in 7, and emphysema in 8. Puncture and aspiration were employed in 2, and the average time of healing was three and one-half to four weeks. Wound of the pericardium occurred in 2 cases. Of the 23 cases in which subcutaneous rupture of the lung occurred without wound of the thoracic wall, the symptoms were as follows: Hemothorax in 3, hemoptysis in 4, pneumothorax in 1, and emphysema in 8. Death occurred in 7, and the average time of healing was four weeks. The complications were: Fracture of the skull in 1, rupture of the liver in 1, and wound of the kidney and hematuria in 1. Of the 7 deaths in the 67 penetrating wounds, in only 2, or at most 3, cases, was the question of operation presented. Of Stuckey's 25 stab wounds, all of which were operated on, death occurred

in 9, abscess of the lung or empyema in 12, and the average time of healing was ten weeks. In Möller's 19 stab wounds which penetrated the lung, none died, in none was there suppuration, and the average time of healing was three and one-half to four weeks. Möller gives the indications for operation as follows: Severe primary hemorrhage; continuing and repeated hemorrhage; severe pneumothorax and emphysema; and secondary pneumothorax. With the observance of these indications, the prognosis of these cases in the future should be somewhat better than they have been up to the present with conservative treatment.

Stasis Hemorrhages Resulting from Compression of the Thorax and Abdomen.—Koch and Ronne (*Archiv f. klin. Chir.*, 1909, xcl, 371) reports a case in which a man was severely compressed in an elevator accident. Immediately after the accident his appearance was alarming. His head and neck were very cyanotic, of a dark blue, almost black, color, and he had small and large petechial hemorrhages under the skin everywhere. The head was swollen out of shape and the breathing was almost imperceptible. The skin of the neck projected over the collar of the clothing, the eyes protruded, and there were subconjunctival hemorrhages. In striking contrast to these phenomena was the slight effect on the general condition. Very often these patients are completely conscious during the whole period of the compression of the chest and abdomen. The cyanosis disappears in a few days, the small hemorrhages somewhat later, the subconjunctival hemorrhages remaining perhaps several weeks. If no complicating lesions are present, in a few days the patients probably feel sound. In 58 collected cases, in only 7 did the complications produce a fatal result. The most probable explanation of the phenomena is that with a closed glottis the blood of the lungs and heart is forced into the peripheral vessels. The cyanosis is very marked in the head and neck and very rare in the extremities, because the jugular veins have no valves. They are occasionally insufficient in the axillary, but are very resistant in the veins of the lower extremities. Disturbances of sight are common, often without demonstrable cause ophthalmoscopically. They were present in 11 (12 with the case here reported) out of the 58 cases. Occasionally there is a brief double blindness, which lasts for some minutes or a half hour. In other cases sight does not return or does so only incompletely, and after some time an atrophic discoloration of the papillæ develops showing that the nerve fibers have become degenerated.

An Experimental and Literary Study Concerning the Manner and Pathway of Extension of Urogenital Tuberculosis.—Sawamura (*Deut. Zeit. Chir.*, 1910, ciii, 203) in investigating the method of extension, assumes that in primary urogenital tuberculosis, the process begins in the kidney and extends through the ureter to the bladder and prostatic urethra, and through the vas deferens to the testicle. It may begin in the testicle and extend in the reverse direction, or beginning in the seminal vesicles, it may extend in both directions to the testicles and kidneys. Upon the basis of the literature it is agreed that the process often begins in the kidney and that the bladder may, in time, be infected, although a sound mucous membrane can, to a certain extent, protect the bladder. Tubercle bacilli in the bladder, usually, will not infect the kidney, if

the normal stream of urine is not obstructed, but will do so in the presence of such obstruction. When tubercle bacilli are injected into the ureter, especially, into the renal pelvis, tuberculosis of the kidney can be produced, with or without ligation of the ureter, although the latter undoubtedly favors its development. The infection must pass by the blood, lymph, or ureter. The blood path is excluded from consideration because by it is produced usually a general tuberculosis. Sawamura carried out experiments on dogs to determine the path of extension. He failed to find that the tubercle bacilli ascended from the bladder through the ureter to the kidney. By direct injection of the bacilli into the lumen of the ureter, without subsequent occlusion of the ureter, a renal tuberculosis was produced. Extending by the lymph paths to the kidney, vesical or genital tuberculosis rarely invades the kidney. It may ascend from the bladder to the kidney without obstruction of the blood stream, when from contraction of the bladder a relatively high internal pressure is produced and in any manner an antiperistaltic movement of the ureter occurs. Tuberculous involvement of the lower end of the ureter may produce the necessary obstruction, stagnation, and dilatation, to permit the tubercle bacilli to reach the kidney. That the process may ascend by the lymph paths cannot be denied, although it has never been established in men or animals with certainty. Tuberculosis of the testicle or epididymis, as a rule, extends through the vas deferens toward the urethra. More rarely it may extend by the lymph vessels. It may remain localized in the testicle and epididymis. Often the vas is involved. The lymph vessels of the testicle go chiefly to the nodes along the inferior cava, near the entrance of the spermatic vein; those from the epididymis to the nodes along the hypogastric vessels. The central (lying next the urethra) portion of the vas deferens, may become infected from tubercle bacilli in the urine. Tuberculous epididymitis can develop from tubercle bacilli in the urine by way of the vas deferens, provided the orifice of the vas or its lumen is blocked, so that with the stagnation of the secretion and exudate, the tubercle bacilli are transported to the epididymis. Extension from a tuberculous epididymis, without participation of the vas deferens, did not occur in Sawamura's experiments, although Oppenheim, and Law and Hausen considered this possible. A primary focus of tuberculosis can develop in the prostrate. It may involve the seminal vesicles and it is assumed, therefore, that it may extend to the epididymis. The seminal vesicles may be involved alone. Sawamura believes that in dogs an ascending tuberculosis of the female genitals can occur.

The Treatment of Bone and Joint Tuberculosis by the X-rays.—Iselin (*Deut. Zeit. f. Chir.*, 1910, ciii, 483) had already obtained excellent results in the treatment of tuberculosis of glands and other soft tissues. Two years ago, at the request of his chief, Prof. Wilms, he undertook the same treatment of the bones and joints. In all, 41 cases were treated, including the bones and joints of the hand, foot, elbow, knee, sacro-iliac joint, and ribs. The method was as follows: In the beginning of the treatment, the bone or joint was exposed to the *x*-rays, three or four times at short intervals, every exposure being made from a different side and always with the fullest dose, until all parts had been exposed. The rays were passed through an aluminum plate, 1 mm.

thick. Because the effects on the skin did not show until two or three weeks, the exposures were made only every three or four weeks. More than three such exposures were unnecessary. From the beginning the joints were placed in a position favorable for cicatricial contraction, except in the case of the small joints. If the tuberculosis healed by cicatrization, after treatment was necessary to reproduce the mobility. This consisted of exposures to hot air, massage of the joints, and movements. This kind of healing was obtained in 10 cases of bone and joint tuberculosis. Almost always the progress was visible, and was obtained in many otherwise hopeless cases. The method is not suited to children, because the epiphyseal cartilage can be damaged, and in the large joints of adults, as the shoulder and hip, the *x*-rays could not be made to penetrate deep enough.

THERAPEUTICS.

UNDER THE CHARGE OF

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Diet in Typhoid Fever.—Coleman (*Jour. Amer. Med. Assoc.*, 1909, liii, 1145) advocates a more liberal diet in the treatment of typhoid fever. He says that the average milk diet of two quarts daily supplies an insufficient number of calories to provide for the increased needs of the body. Consequently patients on a milk diet lose weight and strength and are less able to cope with the disease than patients on a more liberal diet. Coleman advises, as the minimum requirement, a diet containing the equivalent of 41 calories per kilo of body weight. Thus, a man weighing one hundred and fifty pounds will receive during the twenty-four hours a diet equivalent to 3000 calories. Coleman and Shaffer found that the best results were obtained when the diets furnished 60 to 80 calories per kilo. In all instances, the patients on a liberal diet were brighter and stronger and better able to fight the disease. The principal constituents of Coleman's diet are milk, cream, milk sugar, and eggs. In addition, small slices of stale bread or toast, with as much butter as the patient wished, were allowed. He gave to his cases one and one-half quarts of milk, from one to two pints of cream, from one-half to one and two-thirds pounds of milk sugar, and from three to six eggs. Coleman says that he has seen no bad effects from the use of milk in moderate amounts, and he does not believe that it increases the tendency to tympanites. A quart of good milk is equivalent to about 740 calories. Coleman furnishes the bulk of the fat in his diet by means of cream. It is not advisable to give more than one-third of the total calories in the form of fat. A pint of cream contains about 1300 calories. Some of the patients were able to take as much as two pints of cream, but when the larger quantities cause diarrhœa the amount of cream in the diet must be diminished. Carbohydrates protect body

protein better than any other foodstuff. For this reason Coleman supplies a large quantity of the energy of his diet in that form. Starches cannot be used in quantity because of their bulk and the consequent tax on the digestive organs. He prefers milk sugar because it is not very sweet and not so likely to disgust the taste as other sugars, and because it does not so readily produce digestive disturbances. The objections to its use are that in some patients it produces nausea and vomiting, but more often vomiting without nausea. When vomiting occurs, the milk sugar should be stopped. In a few cases milk sugar caused tympanites, but usually the patients could be gradually taught to take and assimilate large amounts. An ounce of milk sugar is equivalent to 120 calories. Milk sugar may be given in the milk; in coffee, tea, or coca, in lemonade, or in custard made with milk and egg. Coleman and Shaffer found that in order to maintain nitrogen equilibrium from 12 to 16 gm. of nitrogen are required in the diet. Approximately 11 gm. are contained in one and one-half quarts of milk and one pint of cream. Coleman supplies the deficiency in nitrogen with eggs. A two-ounce egg will supply 1+gm. nitrogen. The details of administering the diet may be modified to suit the individual case. Coleman gives as a working basis six ounces of milk with two ounces of cream every two hours. From one to four tablespoonfuls of milk sugar are added to the milk and cream mixture. The eggs may be given soft-boiled, poached, or raw in milk with or without whiskey.

Antidiphtheritic Serum and Antidiphtheritic Globulin Solutions.—Park (*Jour. Amer. Med. Assoc.*, 1910, liv, 251) says that until recently the only means of giving diphtheria antitoxin was in the whole serum of the horse in which it had originated. Lately a practical method has been developed to eliminate a portion of the non-antitoxic serum substances while retaining the antitoxin. Park gives a brief description of two globulin preparations containing diphtheria antitoxin. He also points out the fact that the blood serum from different horses varies not only in antitoxic potency, but also in its liability to produce disagreeable after-effects. Thus, different lots of serum of the same manufacturer will vary in liability to produce rashes, and this, together with the idiosyncrasy of the patient, causes some physicians to approve and others to condemn the preparations of the same manufacturers. Park compares the effects of antidiphtheritic serum with those obtained by the globulin preparations. He believes that the globulin preparations contain all the important substances of the whole antidiphtheritic serum. He also states that the rashes and after-effects, in cases observed by him, were undoubtedly much less after the Gibson injections than after the whole serum, and somewhat less after the injections of the Banzhaf modification than after that of Gibson. Curiously enough, only certain types of rashes are eliminated. The urticarial reactions still frequently follow.

Tuberculin Treatment of Tuberculosis.—Löwenstein (*Therap. Monats.*, 1909, xi, 593) used Koch's "old" tuberculin in the treatment of 300 cases of open pulmonary tuberculosis at the Beelitz sanatorium. He commences with a dose of 0.0002 gm., being convinced that smaller doses are liable to induce anaphylaxis. In case of a strong general reaction

with focal phenomena, he waits fourteen or eighteen days before resuming the treatment. After a milder reaction he waits seven to ten days. When the doses of tuberculin have reached 0.1 gm., the intervals between injections should be at least ten days. He does not reduce the dose after a reaction, but increases it more or less according to the intensity of the reaction. Löwenstein terminates the treatment when the patients can stand 0.5 gm. without reaction. In order to avoid a considerable general or local reaction, Koch's "bacillen emulsion" is given instead of the "old" tuberculin. Löwenstein advocates the use of tuberculin in every case in which the physician thinks improvement is possible. He says he has used tuberculin in 1000 cases, and has never observed a dangerous hemorrhage that could be ascribed to the influence of the tuberculin injections. He gives as contra-indications to the use of tuberculin, persistent headache, pointing to the localization of the infection in the central nervous system, nephritis, unless of tuberculous origin, diabetes, epilepsy, and pregnancy.

The Treatment of Gastroptosis.—Von Noorden (*Therapie d. Gegenwart*, 1910, i, 1) believes that the chief indication in the treatment of gastroptosis is to improve the nutrition of the patient. The falling of the stomach is not only a result of stomach atony, but is also due to the lack of support from thin and relaxed abdominal walls. The stomach must never be overloaded, and he advises small and frequent meals of high nutritive value. Solid and fluid food should not be taken at the same time. He advises as an important part of the treatment that the patient should lie down after the principal meals, with the body turned slightly toward the right side. Von Noorden thinks that strychnine physostigmine, and pilocarpine increase the tone of the atonic stomach. He has seen no benefit derived from wearing abdominal binders as regards the position of the stomach, which he determined by the Röntgen rays. However, a binder frequently adds to the general comfort of the patient and is of use especially in nervous patients.

The Treatment of Gastric Disease with Aluminum Silicate.—Rosenheim and Ehrmann (*Deutsch. med. Woch.*, 1910, iii, 111) report their observations regarding the action of aluminum silicate in gastric affections especially those dependent upon a stimulated secretion. They say that in all cases of hyperacidity or hypersecretion of neurotic origin, or associated with organic disease, aluminum silicate acts most favorably in reducing the acidity, quieting the pain, and aiding digestion. Aluminum silicate, as prepared by Kahlbaum under the name neutralon, is a fine, tasteless, and odorless powder insoluble in water. An ideal remedy, they say, should have the power to bind the excessive hydrochloric acid in a harmless combination, and also should have a protective and an astringent effect upon the mucous membrane. They claim that aluminum silicate has these advantages. When taken into the stomach it is broken up by the hydrochloric acid forming silicic acid and aluminum chloride. They state that aluminum chloride has a protective and astringent effect upon the gastric mucous membrane similar to that of silver nitrate and bismuth, without the disadvantage of a possible toxic action. Furthermore, silver nitrate at times causes diarrhœa, and bismuth is constipating. They gave aluminum silicate

in doses of from one-half to one teaspoonful in about three ounces of water one-half to one hour before meals. There were no untoward symptoms from its use. Theoretically they attribute an intestinal antiseptic action to the aluminum chloride and are endeavoring to determine this by further observations.

Substitutes for Digitalis.—Mendel (*Med. Klin.*, 1909, xli, 1551) says that the full benefit of digitalis can only be obtained from preparations containing the mixed glucosides of digitalis. Since the mixed glucosides are responsible for the gastro-intestinal irritation, the only sure way we have of avoiding them is to give the drug intravenously. Digitalin, digitoxin, and digalen do not contain the mixed glucosides, and consequently Mendel has given up their use. He speaks very highly of digitalone, which is prepared from the fresh leaves and accurately standardized. Mendel has given digitalone to more than 200 patients, and has never seen any cumulative action or other untoward effects. The effect of a single dose is not so marked as that of digalen or strophanthin, but Mendel believes it is infinitely safer. Strophanthin and digalen are dangerous because of the tendency to an overstimulation, with consequent depression of the heart. Mendel has seen a large number of patients with marked cardiac insufficiency, who because of their inability to take digitalis internally were kept alive for years by the intravenous use of digitalone. Furthermore, he has found that a single injection of digitalone was often sufficient in cases of acute cardiac failure.

The Treatment of Acute Pulmonary Œdema.—Miller and Matthews (*Arch. Int. Med.*, 1909, iv, 356) base their article upon an experimental research on acute pulmonary œdema. They state that a knowledge of the causes producing an edema is essential to its treatment. Pulmonary œdema is usually a manifestation of some circulatory disturbance. This circulatory disturbance may be due to high blood pressure. In such a case drugs that increase arterial tension are harmful, and so are contra-indicated. The blood pressure should be reduced by bleeding, by counterirritation to the surface of the body, or by drugs that lower the blood pressure. On the other hand, the type of œdema associated with low blood pressure should be treated by drugs raising the blood pressure. They advise against the use of atropine in pulmonary œdema associated with high arterial tension. Atropine is frequently recommended in pulmonary œdema based on its power to lessen secretions. However, the œdema is not due to an increase of secretion, but to a transudation. They believe that adrenalin is probably never useful and often may be dangerous. The inhalation of oxygen is harmless, and often gives temporary relief. Morphine is decidedly beneficial in any type of pulmonary œdema relieving the nervous apprehensions of the patient.

Choral Hydrate as a Local Application.—Heller (*Münch. med. Woch.*, 1909, xlvii, 2418) has used chloral hydrate as a local application in various inflammatory conditions of mucous membranes. He employs a 2 per cent. solution of chloral hydrate in the form of a spray in the treatment of acute tonsillitis. Chloral hydrate has both antiseptic and

anesthetic properties, and is especially useful to relieve pain. He found it of value in the treatment of diphtheria, Vincent's angina, syphylitic ulcerations, and ulcerative stomatitis. When the secretions are foul smelling, chloral hydrate also acts as a deodorant.

PEDIATRICS.

UNDER THE CHARGE OF

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Unusual Persistence in the Secretion of Colostrum.—H. MERRIMAN STEELE (*Archiv. Pediat.*, 1910, xxvii, 32) reports the following case of persistence of the colostrum in a healthy young woman nursing her first child. The baby weighed seven pounds and five ounces at birth and was normal in every respect. It lost steadily in weight, and when it was two weeks old weighed six pounds four ounces. It nursed regularly, seemed satisfied, and had no vomiting or regurgitation. The stools, at first normal, now became greenish and contained some mucus and fatty and proteid curds. There was no fever, and the child slept well. There was scalding about the buttocks, and seborrhœic eczema developed about the face and chest. The mother had a normal convalescence, and her milk was abundant, rich, yellow, and thick. A sample taken for analysis, after standing on ice for ten hours, showed a thick layer of cream resembling butter, the whole specimen being a deep yellow with an olive tint. Analysis showed fat, 3.60 per cent.; proteids, 1.70 per cent.; specific gravity, 1030. As this was not far from normal and the color was peculiar colostrum was suspected. A specimen placed under the microscope showed typical colostrum. The majority of the corpuscles were colostrum bodies and the fat globules varied from minute to exceptionally large size. The baby was taken from the breast and given castor oil. It was weaned on partially peptonized cow's milk and gained four pounds twelve ounces in nine weeks. The skin cleared in eight days. Colostrum continued to be secreted for three weeks, the last examination showing precisely the same condition. In total, the colostrum was secreted thirty-two days, it being fairly assumed that the secretion was colostrum up to the first examination.

Dried Milk as a Food for Infants.—C. K. MILLARD (*Brit. Med. Jour.*, 1910, i, 253) describes the preparation and use of dried milk and the results obtained from its use as an infants' food. Dried milk is prepared by feeding fresh milk in a continuous stream on to revolving cylinders heated by steam to about 250° F., the moisture in the milk being instantly dispelled. A thin film of dry milk forms on the cylinder and is detached by knife-edges. It is subsequently passed through a sieve and is obtained as a coarse, granular, cream-colored powder practically sterile, which, in air-tight packages, will keep almost in-

definitely. The relative proportions of the main constituents—proteids, fats, milk sugar, and salts—remain practically unchanged, but changes occur in the more complex albuminoids and enzymes, similar to those in boiled milk. The extremely short time during which the milk is subjected to the heat by the Just-Hatmaker process described above, may cause less change than occurs in boiling. When mixed with water, about 60 per cent. of the dried milk is soluble, the remainder is readily suspended. This dried milk has been used at an Infants' Milk Depot for eighteen months, for about two hundred infants. One advantage discovered was greater digestibility; many infants with whom liquid milk did not agree, thriving on the dried form, and retained it. This difference is accounted for by the character of the curd formed in the stomach which does not tend to form hard cheese-like masses. All infants not thriving on bottled milk were placed on the dried milk, with excellent results. After a period of ten months or longer careful records and investigations showed no scurvy or rickets resulting from its use and no bad after-effects have been discovered. The advantages of the dried milk appear to be: *Ease of digestion, bacterial purity*—freedom from tubercle bacilli and contamination by flies. *Conservatility*—no "souring" in hot weather. *Convenience*—a definite quantity being mixed with warm boiled water. *Cheapness.* The presumed destruction of the antiscorbutic properties of the milk is theoretical, but can be compensated for, if thought necessary, by administering fruit juice. Dried milk, being after all "only milk," is in an entirely different category from all patent foods prepared from cereals, and is superior to them.

Cyclic or Recurrent Vomiting with Hypertrophic Stenosis of the Pylorus.—A. E. Russell (*Brit. Jour. Children's Dis.*, 1910, vii, 49) supports the argument that muscular spasm of the pylorus due to hypertrophic stenosis is sometimes the cause of cyclic vomiting with its attendant conditions. He cites as an example the case of a boy, aged four years, and nine months, who from birth was subject every few months to attacks of vomiting with epigastric pain. The attacks appeared suddendenly and lasted twenty-four hours, the vomiting recurring during the day. The child's last illness began with an attack lasting one week, during which the vomitus turned from yellow to coffee color. There was great prostration, emaciation, and the breath smelled strongly of acetone. There was constipation and the urine contained acetone and diacetic acid. Then a period of remission occurred lasting nineteen days, during which the child ceased vomiting, took nourishment and improved. The urine became free of acetone and diacetic acid. There was then a return of the vomiting and epigastric pain and after five days the child died, acetone and diacetic acid again having appeared in the urine. The autopsy showed a considerably dilated stomach. The lumen of the pylorus was very small and its walls were thickened. There was no ulcer or scar tissue present and the remaining thoracic and abdominal organs were normal. These symptoms are practically identical with those of cyclic or periodic vomiting in children. The current views as to cyclic vomiting are that it is due to a poisoning arising from the intestinal tract, with imperfect oxidation of fats and an accumulation of them in the liver. Russell argues that acute starvation accompanies this condition, as evidenced by the emaciation and the

acetone bodies in the breath and urine (with the fatty changes in the liver often found in these cases). He claims that these latter conditions can be explained by the acute starvation involved with cyclic vomiting, and that the cause of the vomiting is elsewhere, probably in the hypertrophic stenosis of the pylorus. While actual stenosis of the pylorus is not an essential factor in the disease, he submits that the attacks were due to the occurrence of pyloric spasm. On this hypothesis, as long as pyloric spasm lasted obstruction would be complete. If it persisted long enough, acute starvation would necessarily follow with the resulting acidosis. Fatal issue followed on the inanition and exhaustion. While possibly not a factor in all cases, pyloric spasm is enough to account for recurrent attacks of vomiting and presents all features described as characteristic. It is also consistent with the fact that the attack often comes on suddenly. Relaxation of the spasm would be followed by this sudden cessation of the attack, which is often a noticeable feature.

An Epidemic of Acute Poliomyelitis.—W. W. Treves (*Brain*, 1909, xxxii, 28) records the occurrence of an epidemic of 8 cases of acute anterior poliomyelitis in Upminster, a town of 1700 inhabitants. It was the first epidemic of its kind in the town, and no case of infantile paralysis had occurred there in several years. The months of the epidemic were hot and dry, but the heat was not excessive. Six of the patients had constitutional symptoms and a few days afterward were paralyzed; one child had fever, but developed no paralysis; the eighth was paralyzed without any constitutional symptoms. The legs were the members most commonly affected. In some of the children the eyes attracted the parents' attention by their peculiar look, but in no case was any definite evidence of polio-encephalitis obtained. Seven of the children were over six years of age, one was three and one-half. In 5 of the cases the period of incubation could not have been more than six days. All attempts to trace the means by which the disease spread failed. Of 32 other epidemics recorded in literature and discussed by the author, but 2 occurred in England.

The Dwarf Tapeworm, an Intestinal Parasite in Children.—Oscar M. Schloss (*Archiv. Pediat.*, 1910, xxvii) reports 14 cases of dwarf tapeworm or Hymenolepis nana, in 230 children. The average length of the worm is from 14 to 16 mm. The distal half is broad, while the proximal half becomes narrow. The segments are from 3 to 6 times as broad as long and the head of the worm is globular and carries four suckers and a rostellum armed with twenty or thirty bifid hooklets. Its habitat in man is in the upper two-thirds of the ilium. The eggs are slightly oval and have two membranes widely spaced. From the poles of the inner membrane are projections from which spring filaments which ramify in the space between the membranes. This is characteristic. The 230 children examined were from the tenement-house district, and, with one exception, were all born in New York City. Six of the 14 cases observed showed no symptoms referable to the parasite. The remaining 8 cases showed gastro-intestinal and nervous symptoms. Under the former, epigastric pain, nausea, vomiting, and an increased appetite were prominent. Restlessness at

night, grinding the teeth, itching of the nose, and genital pruritus under the latter. Eosinophilia was present in 7 of the 8 cases suffering from symptoms of the parasite. In cases with no symptoms eosinophilia was uniformly absent. A secondary anemia was generally present. The absence of, and variety in, symptoms are probably due to the site of mechanical irritation in the intestine and to toxic effects. The mode of infection is through ingestion of the ova in food. No intermediate host has been found in any human food. The dwarf tapeworm, however, has frequently been found in the small intestine of rats. Autoinfection is possible, owing to the great number of ova in the feces. The diagnosis is made by finding the characteristic ova in the feces or by obtaining the parasite after treatment. The treatment consists of a preliminary period of two or three days on liquid diet, a preliminary purge and the administration of oleoresin of male fern in mixture, emulsion, or capsule. The dose for a child two to four years old is 0.5 dram; four to six years, 40 grains; and six to twelve years, 1 dram. This is given on an empty stomach. It is divided into three or five doses and given at half-hour intervals. A brisk cathartic is given a half an hour after the last dose is taken. When the treatment is not effective the ova reappear in the feces in fifteen days.

OBSTETRICS.

UNDER THE CHARGE OF
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The Diagnosis of Puerperal Septic Infection.—Sachs (*Zent. f. Gynäk.*, No. 46, 1909) gives the result of 200 examinations of lochial discharge and blood in septic cases. This study was made to determine the significance of hemolytic streptococci in the blood as well as in the lochial discharges. He agrees with Veit that serious puerperal septic infection is caused by these organisms, which are present in the great majority of cases. By using fluid blood agar media he was able to recognize hemolytic streptococci in two-thirds of the cases. It is not sufficient to recognize a few of these organisms to make a diagnosis of infection. Their presence must be sought in the blood and their frequency estimated. When puerperal ulcers with hemolytic streptococci are present the prognosis is better than if peritonitis has developed. Recognition of hemolytic streptococci in healthy puerperal patients has absolutely no significance with regard to their importance in cases of sepsis. When these germs are not found in a septic patient, the prognosis is good. As an exception to this, are those cases late in the puerperal period in which hemolytic streptococci have passed from the uterus and have caused suppuration in thrombosed veins, and are no longer recognized in the secretion of the uterus; also in cases of sinus thrombosis and other intercurrent affections in which the streptococcus is the active agent.

In cases of mild infection in greatly weakened persons after severe hemorrhage or asphyxia following anesthesia, a fatal result may follow, although hemolytic streptococci are not found. When peritonitis develops early in the puerperal period perforation of the uterus must be suspected, and in these cases hemolytic streptococci might not be obtained from the uterine cavity. The mortality statistics of surgical operations in streptococcic peritonitis give 50 per cent. recoveries, and 50 per cent. deaths. This favorable showing is to be explained by the diminished virulence of the germ, and the fact that many of these cases are perforation of the uterus. The high mortality of severe puerperal sepsis arises in great part from the fact that a differential diagnosis between the mild and severe cases is not made sufficiently early to be of use in the treatment. Clinical observation will often determine the degree of severity in septic infection, but bacteriological examination is a most useful adjunct.

Modification of Peripheral Sensation during Pregnancy.—Pondolfi (*Annali di Ostetricia et Ginecologia*, No. 9, 1909) contributes a paper upon this subject, describing an apparatus which he has devised for testing the peripheral sensibility of patients during pregnancy. His experiments were made upon the fingers, and in all 30 cases were studied. He concludes that peripheral sensibility to pain during gestation is very considerably decreased.

Ovariotomy and Myomectomy Early in Pregnancy, with Full Term Delivery.—Grad (*Jour. Amer. Med. Assoc.*, November 27, 1909) reports the case of a patient in her first pregnancy brought to the hospital because she had fainted on the street, after complaining of sudden abdominal pain, with vomiting and collapse. This pain gradually subsided, leaving the abdomen tender. There was a history of cramp-like pain in the abdomen, with moderate fever, indigestion, and disturbance of the bladder, for a week or ten days prior to this attack. The patient had been married nine years, but had not previously been pregnant. On examination the uterus was slightly enlarged, with several fibroid nodules. A large movable tumor was also detected in the pelvis. The diagnosis of ovarian cyst with twisted pedicle and pregnancy in a fibroid uterus was made. At operation the pedicle of the cyst was ligated and the tumor removed. Three fibroids were enucleated without especial difficulty. Although the patient had a bloody discharge from the uterus after the operation, the ovum was retained, and the patient went to term and was subsequently delivered by the use of forceps.

Ovarian Cyst with Twisted Pedicle Complicating Pregnancy.—Rushmore (*Surg., Gynecol., and Obstet.*, November, 1909) reports the case of a multipara, who on the day before her admission to the hospital had cramp-like pain low down on the left side. Examination under chloroform revealed pregnancy with an ovarian tumor. On opening the abdomen an ovarian cyst on the left side with the pedicle twisted one and a half times, dark purple, almost black in color, was found. The tissue was very soft and friable, and the wall of the uterus bled freely. The tumor was successfully removed, and the mother made a good recovery,

a healthy child being born at full term. On examination the tumor was a dermoid cyst of the ovary with strangulated pedicle and a partial strangulation of the Fallopian tube. The article concludes with a review of the literature of the subject.

Artificial Reproduction of the Amniotic Liquid during Labor.—Schallehn (*Archiv f. Gynäk.*, 1909, lxxxix, Heft 2) reports five cases of premature escape of the amniotic liquid, in which Bauer's elastic bag was introduced, distended with salt solution, and allowed to remain in place in the membranes. In several cases in which the heart sounds had become weakened through birth pressure they improved after the bag was introduced. If the patient suffered much pain from pressure, morphine was given hypodermically, and the patient was delivered so soon as the cervix was dilated by version or forceps. The presence of the bag seemed to excite uterine contractions and lessen the risk of fatal birth pressure for the child. It was used in these cases, not primarily to dilate the cervix, but to protect the child from pressure; secondarily to soften the cervix and expedite labor.

The Results of Pregnancy Occurring After Operations for the Correction of Retroflexion.—Birnbaum (*Archiv f. Gynäk.*, 1909, lxxxix, Heft 2) reports the results in 20 cases operated upon for retroflexion by ventrofixation. In 4 of these pregnancy occurred, terminating in labor without complications. In these cases there were evidences of peritoneal adhesions and alterations of the tubes and ovaries. These were detected at the operation. The cause of the sterility which had existed before operation seemed to be the kinking in the Fallopian tubes, which was caused by the retroflexed condition of the uterus. In 3 cases no cause could be found at operation for the peritoneal adhesions; in 1 case a previous parametritis had undoubtedly existed. In the 16 other cases in which operation was done for retroflexion, pregnancy had not occurred at the time of writing. It is questionable whether lesions indirectly produced by the retroflexion were not responsible for the sterility in these cases.

GYNECOLOGY.

UNDER THE CHARGE OF

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An Ovarian Abscess Containing a Lumbricoid Worm.—Fry (*Jour. Amer. Med. Assoc.*, 1909, liii, 1028) reports a case of ovarian abscess that contained a lumbricoid worm. The patient was twenty-three years of age. The right appendage was inflamed and adherent. The left ovary and tube were adherent to the uterine cornu. The ovary was enlarged to the size of a hen's egg, the surface smooth and

non-adherent to intestine. It ruptured during removal and 30 c.c. of pus escaped into the abdominal cavity. Projecting from the abscess through the rupture was a lumbricoid worm 6 or 7 cm. in length. It was dead and flattened. The worm was identified by Dr. B. H. Ransom of the United States Department of Agriculture. The pus contained Bacillus coli communis in pure culture. Fry concluded the worm had gained access to the ovary by the vaginal route and entered the ovary through a ruptured Graafian follicle.

The Choice of Operations for Retrodisplacements of the Uterus.—Benjamin (*Jour. Amer. Med. Assoc.*, 1909, liii, 1072) states: Retrodisplacements of the uterus often cause much discomfort. The harmonious action of all the supports is essential to the uterus for its normal position. The operation which interferes with the laws governing the normally placed uterus is not to be advocated. The operation which produces unnecessary intra-abdominal traumatism should not be chosen in the ordinary case. Operations which could possibly interfere with the enlargement of the uterus during pregnancy should be used in selected cases only. Operations which leave an additional suture line within the abdomen may cause subsequent trouble. Operations which do not give as strong a support as possible consistent with the normal functions of the uterus may result in failure in some cases. The operation which utilizes the normal ligaments with little or no traumatism is less troublesome and more scientific. Benjamin then describes his modification of Gilliam's operation for shortening the round ligaments and gives the advantages of it.

The Endometrium and Some of its Variations.—Gardner and Novak (*Jour. Amer. Med. Assoc.*, 1909, liii, 1155) deprecate the employment of many terms in quite common use, that are now known to have been coined from mistakes in pathology. They believe Hitschmann and Adler have taken an extreme view in practically asserting that glandular changes do not occur except in connection with the menstrual process. Both animal experimentation and clinical observation indicate that the actual underlying cause of menstruation is the secretory activity of the ovary, which produces an internal secretion or hormone essential for its occurrence. The principal effort of this substance seems to be of a vasomotor nature, and is exerted especially on the pelvic bloodvessels. It is only natural to suppose that the endometrium plays a purely passive role in this phenomenon, and that the histological changes observed in connection with menstruation represent merely the reaction of the endometrium to the process—a reaction which may, however, be elicited by influences other than that of normal menstruation.

Factors which Contribute to a Reduction in Mortality in Abdominal Surgery.—F. F. Simpson (*Jour. Amer. Med. Assoc.*, 1909, liii, 1173) discusses in detail the factors contributing to a minimum mortality rate in abdominal surgery. While it is a paper not amenable to being satisfactorily abstracted, his conclusions may be considered as follows: An accurate knowledge of the nature, extent, and kind of disease, and exact determination of the patient's margin of reserve strength; a judicious adaptation of the time and type of operation to

individual needs; a group of competent operative co-workers; a minimum amount of anesthetic; a rigid aseptic technique; and speed with precision, are factors which will yield a low mortality and highly satisfactory operative results.

The Age of Menstruation in Egyptian Girls.—Mrs. B. Sheldon Elgood, Assistant Medical Officer, Ministry of Public Instruction, Cairo (*Jour. Obst. and Gyn. of Brit. Emp.*, 1909, xvi, 242) has studied the subject of the date of first menstruation in Egyptian girls, her field of inquiry being several large schools for native Egyptian girls. In 83 menstruating girls, the birth certificates of whom were available, she found the first appearance of menstruation in 12 was at twelve years; in 44, at thirteen years; in 21, at fourteen years; and in 4, at fifteen years. This study tends to prove that at thirteen and fourteen years 80 per cent. of native Egyptian girls begin to menstruate.

The Anatomy of Tubal Convolutions and the Mechanism of Tubal Occlusion.—James Young (*Jour. Obst. and Gyn. Brit. Emp.*, 1909, xvi, 307) states that his analysis of the various theories advanced to explain the disappearance of the tubal fimbriæ reveals the fact that they fall under one or other of two headings: (1) The first class includes the theories, which explain the process as being due to an increase in the total length of the tube wall, which, by expanding in an outward direction, becomes projected beyond the tubal fimbriæ. According to the theory of Alban Doran, which receives the support of Kleinhans, the increase in length is dependent on the swelling and increase in substance of the tube wall associated with salpingitis, etc. According to Emil Ries the gliding outward of the "peritoneal ring" over the fimbriæ is rendered possible by the fact that the walls become loose and redundant subsequent to the collapse of a distended tube. (2) In the second category are included the theory of Opitz, which explains the process as due to retraction of the muscular and mucous coats of the tube within the serous coat, and the theory described in this paper, in which the gliding process involves only the mucosa and inner coat of muscle. In the so-called "perimetritic closure" of Alban Doran the sealing of the opening is explained by a matting together of the fimbriæ by inflammatory adhesions without a preliminary recession.

Removal of an Unusually Large Ovarian Tumor.—Knight (*Amer. Jour. Obst.*, 1909, lxi, 441) reports the successful removal by abdominal section of an ovarian cyst weighing one hundred and eleven pounds. It had been observed for ten years by the patient and was removed without preliminary aspiration.

Enucleation of Uterine Myomas; Why and When Performed.—Montgomery (*Jour. Amer. Med. Assoc.*, 1909, liii, 1245) suggests the following conditions as indicating hysteromyomectomy: (1) When the growths are few in number and the structure of the uterus is but little involved. Of course, the fibroids may be numerous but situated so near the surface as to permit their removal with but little injury to the general structure, but the large number indicates a tendency to fibroid degeneration which presages early redevelopment. When a number

of growths of considerable size are present, the structure of the uterus is so spread out and will be so injured as to render an attempt to save the organ attended with danger during the subsequent convalescence and an element of danger in the event of pregnancy and labor. (2) When the growths are readily accessible through the vagina or cervical canal. A growth within the uterus, either a sessile, submucous, or an interstitial, is readily attacked. Not infrequently, the canal may be partially dilated and the dilatation can be completed by the introduction of tents, or the cervix may be split bilaterally until the tumor is exposed or rendered accessible. The enucleation completed, the cavity may be packed with gauze and the split cervix closed much as is done in an ordinary trachelorrhaphy. The vaginal operations are attended with less constitutional disturbances than in the removal by an abdominal incision. (3) When the woman, whether unmarried or married, is under forty years of age, and particularly when she is childless or has but one or two children. The removal of the growths at an earlier period cannot be considered as rendering certain the escape of the patient from recurrence, for one of his patients who had two fibroids enucleated when she was thirty-three years old, five years later had twenty removed. The age of forty, however, is one at which the individual suffering from such growths begins to undergo retrogressive degenerations, and when the patient has not previously been fertile pregnancy is much less likely to occur. (4) When the tubes and ovaries are free from complicating conditions. The existence of tubal or ovarian disease of sufficient gravity (as hydrosalpinx, or pyosalpinx, or ovarian hematoma), to render the probability of conception remote or to necessitate the removal of tubes and ovaries to insure restoration of health, should also be an indication for the removal of the fibromyomatous uterus. While it is true that in the majority of cases the tumors decrease and become quiescent after the menopause, yet they sufficiently often undergo necrosis and other degenerative changes to justify the removal of the uterus.

OPHTHALMOLOGY.

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Treatment of Detachment of the Retina.—DEUTSCHMANN (*Ophthalmoscope,* November, 1909, p. 737), in demonstrating his methods of operating for this condition, bisection and injection of the sterile vitreous humor, formulates the following rules: Bisection: never operate upon a recent detachment so long as the detached part is situated in the upper part of the fundus; the bisection is to be made with a double-edged linear knife *downward* in the anterior boundary of the cul-de-sac.

Bisect horizontally, guide the knife tangentially to the eyeball from downward and outward to downward and inward. Make the bisection as quickly as possible in a straight direction through the eyeball avoiding the junction at the spot of the counter puncture, and draw back the knife in the same way it was introduced. Turn the blade a little at the spot of the puncture, so that the retinal and eventually the preretinal fluid can escape. The operation can be repeated twenty times or oftener unless interference has been followed by any unfavorable result. Bandages should be applied to both eyes for the first twenty-four hours and then only upon the operated one for four or five days. Atropine should be employed during the entire treatment and the patient kept in bed for a week after each operation. The injection method is reserved for cases otherwise hopeless.

Myopia and Light Sense.—Landolt (*Klin. Monatsbl. f. Augenhk.*, October, 1909, p. 369) concludes that the light sense is not influenced in myopia even of high degree unless decided chorioretinal changes are present, and even the latter do not always diminish that function; neither does astigmatism have any effect, and light sense and visual acuity are independent of each other. Age, however, appears to diminish the faculty in myopes as well as in emmetropes and hyperopes.

Report upon 103 Cases of Magnet Extractions.—Hausmann (*Klin. Monatsbl. f. Augenh.*, 1910, xlvii, 86) reports that of 103 magnet extractions from the ophthalmic clinic of the University of Halle, the vision ranged from $\frac{5}{5}$ to $\frac{5}{15}$ in 37 cases; from $\frac{1}{3}$ to $\frac{1}{30}$ in 11 cases; and less than $\frac{1}{30}$ in the same number; in 15 cases the form of the eyeball was maintained, though the vision was lost; in 7 there was phthisis bulbi, and in 22 enucleation or evisceration had to be performed.

Etiology of Subacute and Tardy Infection Following Operations.—(Ophthalmic Section of XVIth International Congress of Medicine, Budapest, *La Clin. Ophtal.*, November 10, 1909, p. 567). Following a lengthy discussion upon the infectious complications which sometimes follow iridectomy, extraction of cataract, discission, sclerotomy, and other operations upon the cornea, iris, uveal tract, or vitreous body, Morax comes to the following conclusions: The tardy appearance of an iridociliary infection of subacute development can be provoked by a late development of pyogenic microbes, which have been introduced at the time of the operation. Although bacteriological examinations are still liable to be misinterpreted and while the explanation of the majority of such cases of iridocyclitis is purely hypothetical, the reporter is inclined to believe that they are in general due to the development of little known saprophytes and still undescribed spores which have their seat upon the surface of the conjunctiva of certain individuals. These germs offer to the usual methods of disinfection of the conjunctival cul-de-sac greater resistance than the ordinary pyogenic microbes. At the same session Angelucci considered postoperative inflammations caused by auto-infection. Senile and arthritic albuminuria occasion no interference with the cicatrization of wounds; grave forms of Bright's disease, however, frequently give rise to iritis. Neither does diabetes, save in its graver forms, interfere with the healing process. Gout

introduces no complications except when there is also disturbance of the intestinal tract. Postoperative iritis frequently appears in connection with dental suppuration, constipation or intestinal infection, and occasionally also in vesical catarrhs. Influenza may provoke endogenous suppuration in an eye recently operated upon, and so may furunculosis and abscesses, no matter where situated.

Nervous Asthenopia from Electric Light; Use of Yellow Glasses.—De Waele (*Archiv. d'Ophtal.*, September, 1909, p. 566) publishes six instances in young persons in whom asthenopia was produced by working under arc lights. While the electric light may be no richer in ultra-violet rays than solar light, the former is more dangerous because the eye is more directly exposed. Electric lights should be provided with glass globes (yellow is the best), or at least so placed or screened that the eyes shall be protected from the direct rays. When this can not be done yellow glasses should be worn.

Trachoma in the Abruzzi, Italy.—Guiseppe's (XIth International Congress of Ophthalmology, *Rec. d'Ophtal.*, August, 1909, p. 255) statistics show what ravages trachoma causes in that country; in a population of 147,000, more than 2000 cases of trachoma are known. The disease is especially common in the valleys, the mountains being almost exempt. In many communities the malady has been imported by Italian emigrants returning from Brazil.

Subcutaneous Injections of Alcohol in Blepharospasm and Spastic Entropion.—Fumagalli (*Annali di Oftal.*, 1909, xxviii, fasc. 3, p. 162), at the Clinic of Turin, makes the injections superficially in the neighborhood of the stylomastoid foramen under the skin, in the region of the supra-orbital nerve and of its palpebral filaments and in the distribution of the orbital filaments of the facial so as to affect the orbicularis. Thirty parts of absolute alcohol and 60 of sterilized water is the injection employed without an anesthetic. A syringeful (Pravaz) is used for the supra-orbital region, and in inveterate cases of essential blepharospasm a similar quantity is employed for the infra-orbital region. A single injection under the skin of the lid, in the centre and parallel to the free border, suffices in spasmodic entropion (children and the aged). Several injections may be made daily or at longer intervals until cure or considerable amelioration is obtained.

Helmholt's Theory of Accommodation.—Roche(*Rec. d'Ophtal.*, October, 1909, p. 325) observed a case of complete bilateral ectopia of the lens. The aphakic portion of the pupil was hyperopic 10 D.; the portion opposite the lens was myopic 13 D. This case and others like it furnish a conclusive argument in favor of Helmholt's view that during accommodation the zonula is relaxed, against the opinion of Tscherning that the act of accommodation is brought about by tension of that membrane. The fact that increase of the refraction is not always observed in luxation of the lens may be due to the circumstance that the fibers of the zonula are not completely torn through—there is subluxation, a comparatively small number of fibers being sufficient to maintain the shape of the lens.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF
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The Nature of Antitrypsin in the Blood Serum and its Mode of Action.—Pick and Pribram have shown that when the blood serum is treated with ether it is robbed entirely of its antitryptic qualities. This naturally suggests that the antifermentive property of the serum is in some way dependent upon the presence of lipoid substances. O. SCHWARZ (*Wien. klin. Woch.*, 1909, xxii, 1151) has reported and confirmed these experiments, and in investigating the subject still further brings out many points of interest. He has found that 5 per cent. emulsion of lipoid will inhibit, though not as powerfully as the same quantity of blood serum, the proteolytic action of trypsin. Blood serum which has been inactivated by extraction with ether may be re-activated again by the addition of amounts of lipoid emulsion, not in thenselves markedly antitryptic. The re-activation does not take place, however, unless the lipoid and serum are allowed to remain in contact for one hour at 65° C. It seems, therefore probable that the lipoids must form a combination with albuminous substances of the serum in order to assume an antitryptic power. It could further be shown that when this albumin-lipoid complex is brought in contact with a solution of trypsin a portion of the trypsin is actually used up and is probably bound to the inhibiting substance. As far as could be learned the antitryptic and antipeptic properties of the serum are not identical, for when the serum was inactivated by ether extraction for trypsin it was still active against pepsin. Finally, in a few isolated experiments, it could be shown that the antitryptic property of the serum increased in proportion to the amount of lipoids present. Many observations have been made upon the antitryptic and antileukoproteolytic power of the serum in various diseases, and this property has been found to vary widely, but a number of observations seem to show that an increase in the antitryptic and antileukoproteolytic property accompanies an increase in the number of white cells. This Schwarz believes is due to the destruction of cells with subsequent liberation of lipoids and not, as has been suggested, to the formation of a true antiferment in the sense of an antibody, through the liberation of ferment substances in the blood.

The Venous Pulse under Normal and Pathological Conditions.—RIHL (*Zeit. f. Exp. Path. und Ther.*, 1909, vi, 619) discusses extensively from an experimental point of view the mechanism of the venous pulse, and his work must be the final word on a number of points. In his experiments 133 dogs were used, and observations were made with the

thorax both opened and closed. He found that the placing of a funnel over the pulsating parts gave more delicate results than when a manometer was used. The three principal venous waves, the *a*, the *c*, and the *v* waves are discussed separately. Rihl concludes that the *a* wave is caused entirely by the auricular systole. An actual column of blood is sent up into the vein by the auricle and the pressure of this column carries the wave above the intact vein valves. It is not a passive or congestive wave. The *a* wave is increased by increase of auricular systole, auricle and ventricle contracting simultaneously, and by venous engorgement. The latter cause may increase the wave even when the auricular systole decreases. The *a* wave is diminished by a decrease of auricular systole, and from this cause it may disappear. The ventricular activity causes two venous waves, the *c* wave and the *v* wave. These occur when the ventricle contracts without the auricle. The *c* wave is not dependent on the motion of the aorta or carotid artery. The *c* wave and the carotid pulse are synchronous when funnels are used on both sides, and the *c* wave, which follows a little after the systolic contraction, is not effected by the presence or absence of the auricular systole. When the venous wave is taken from a deeply inserted cannula in the heart the *c* wave is synchronous with the ventricular systole. The relation between the *a* wave and *c* wave depends, in part, on the time between the *a* and *v* systole, but is also dependent on the size of the *a* wave. Too much dependence should not, therefore, be put in this relation in determining the state of the conduction of the heart beat. The *v* wave commences during ventricular systole, and this fact shows that it is not dependent on ventricular diastole for its formation. This wave is best considered as formed by two forces, the engorgement of the vein during ventricular systole, and the movement of the base of the heart upward at the beginning of diastole. When a division occurs in the *v* wave corresponding to the point where the two forces meet, it is synchronous with the dicrotic notch, as seen in the carotid artery tracing. This division represents, therefore, the opening of the atrioventricular valves. This *v* wave is increased and decreased with the increase and decrease of venous engorgement. Slight tricuspid lesions cause no changes in the *v* wave, but grave lesions increase it and make it come earlier in systole. Only with the highest grade lesions does the ventricular type of venous pulse occur.

The Cause of Arteriosclerosis.—Harvey (*Virch. Archiv*, 1909, cxcvi, 303), attempting to discover what part increased blood pressure might play in the production of arteriosclerosis, has compressed the abdominal aorta of young rabbits for three minute periods over a prolonged time. By actual manometric tracing it was found that digital compression of the aorta in rabbits raises the blood pressure at times 42 mm. It was found that by this method extensive sclerosis could be produced in the aorta above the point of compression. The sclerosis was of the type described by Monkeberg in man, and consisted in degeneration of the muscular coat with deposits of lime salts. Harvey believes that the sclerosis produced in rabbits by injection of adrenalin, nicotine, etc., is caused not by a toxic action of the drugs, but by their power to increase blood pressure.

Changes in the Chromaffin System in Cases of Unexplained Postoperative Death.—Joseph Hornowski (*Virchow's Archiv*, 1909, cxcviii, 98) points out that sudden death after operation, with symptoms of shock, has been explained by hypotheses only—*e. g.*, chloroform, heart failure, etc. He argues however, that this is not correct and that such definite clinical symptoms as these cases present must have an equally definite cause. This cause he attempts to show lies in the so-called "phaochrome" or chromaffin cells of the adrenal glands and sympathetic ganglia, and brings forward as an analogy the extreme asthenia of Addison's disease in which these cells show marked change. This change is a loss of brown color when stained by chrome salts In four cases of death shortly after operation he found the phaochrome cells of the adrenals and sympathetic ganglia either colorless or only very faintly yellow. From these and other unreported observations Hornowski concludes that the pale appearance of these cells is the sign of a lack of activity on their part, or a lack of the "pressure-maintaining substance" which they produce and is, therefore, sufficient to explain death in the absence of other causes. With this hypothesis as a starting point the author reasons that the blood-pressure-lowering effect of chloroform is offset by the secretions of the chromaffin cells. This extra call upon the cells tends to exhaust them, but in addition the drug exerts a toxic action upon them, so that a point is reached where the cells are no longer able to meet the vital demand and death ensues. As suggestive corroborative clinical observations the author mentions the occurrence of death in those cases in which the patient passes through a long period of excitation in the first stages of anesthesia and consequently uses a greater amount of the "pressure-maintaining substance." Furthermore he cites those patients that feel unduly well and bright immediately after operation and then go on to sudden death. This stimulated condition he believes is a manifestation of excessive production of the substance, and the rapid subsequent collapse evidence of exhaustion of the chromaffin cells. Hornowski then undertook animal experiments. He anesthetized rabbits for various lengths of time and also injured the sympathetic ganglia. He found that short deep chloroforming produced no change in the phaochrome cells. Repeated, long chloroforming, however, caused the cells to fail to take the chrome stain. Trauma, on the other hand, to the peritoneum, adrenals, and sympathetics, produced rapid loss of color in the cells of the chromaffin system. The author concludes that the brown color (chrome reaction) is an indication of the power of the cells to produce the "pressure-maintaining substance," and that if the organism can meet the increased demand for this substance caused by chloroform and trauma—in the face of the toxic effect of the chloroform—death does not occur.

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